DISEASES OF THE EAR
IN CHILDHOOD

BY

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VIENNA

TRANSLATED BY

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Preface

Almost every year new text-books on Otology are published, descriptive of the rapid progress and status of this branch of medical science. The pathological conditions which occur in infancy and childhood are described briefly, but there is no characteristic and exhaustive description of the anatomical, clinical, and therapeutic peculiarities, which are important factors in the child's ear.

The publication of the present volume was prompted by a desire to fill this need, and I willingly complied with the request of the editors of "The Diseases of Children" to write a supplementary volume dealing with the affections of the ear in infancy and childhood.

The many peculiarities of childhood impart a special character to the affections of the ear, and give rise to pathological pictures peculiarly their own.

The consideration of this fact was a sufficient incentive to undertake the work, but other factors rendered its execution imperative, namely, the great frequency of oral affections in infancy and childhood, and their importance in the mental and physical development of the young.

It was difficult, however, to decide upon the amount of detail in which the various sections should be presented, for, if this volume were to adapt itself to the requirements of the pediatrist in accordance with the former ideas on the subject of juvenile otology, it would have been necessary to condense the available material. This, however, would have materially interfered with the scientific treatment of the questions involved.

Feeling that the requirements of the modern pediatrist would not be served by a simple compilation of therapeutic indications, I decided to include all anatomical, clinical, and diagnostic facts, aside from the therapeutic armamentarium, which represent the present knowledge of our specialty. Bearing the practical importance of the latter in mind, I have endeavored to satisfy the demands of the general practitioner, so that he may obtain a clear understanding of the pathological pictures, of their causes and development, of the therapeutic measures to be adopted, and of the correct interpretation of difficult and complicated conditions which should be referred to the otologist for rational treatment. It need not be specially emphasized that a correct procedure on the part of the attending physician is of extreme importance in otological complications.

The child's ear differs materially from that of the adult in various particulars, which are not only of theoretical but also of clinical importance, and which for purposes of diagnosis and treatment should not be neglected. For this reason the anatomy of the child's ear had to be described in detail. At the same time, this chapter has been so arranged as to facilitate the study of the text when comparing it with anatomical preparations.

In regard to the physiology of the ear, I have confined myself to the most necessary details. The theory of hearing and the physiology of the vestibule have only been discussed in so far as is necessary for a clear understanding of the clinical methods of functional tests.

In the section on clinical methods of examination, I have described those which are in use in my own department, where I have always attached the greatest value to combining exact methods with rapid and convenient application.

In the arrangement of the clinical material, I have followed the method I am in the habit of using in my clinical lectures. In discussing the affections of the labyrinth and the extra- and endocranial affections of the ear, I have selected that method of grouping which has proved to be necessary in keeping step with the progress of modern otology. The oral affections of infancy have been given special consideration.

The increased interest which has been extended to juvenile oral conditions in modern times has been followed by astonishing results in the education and care of the deaf-mute. Accordingly, it was necessary to discuss deaf-mutism, the education of the deaf-mute and those with difficult hearing, as well as the question of school physicians. Deafness in cretins, endemic deafness, and constitutional oral affections have been treated in a special chapter. The book also deals with a number of oral affections which, up to a short time ago, were regarded as diseases of the adult; they include various forms of labyrinthine deafness and otosclerosis, the foundation of which may have been laid in childhood or *in utero*, as has been shown by anatomical findings.

Diseases of the ear occurring in conjunction with infectious diseases are only mentioned, so far as their characteristic facts are concerned, while the details of the pathological pictures and the local treatment are dealt with in preceding chapters.

The most important affections to be discussed are those of the middle ear, especially suppurative inflammation and its complications. The extra- and endocranial otogenous affections are, therefore, arranged as independent sections attached to other chapters. Only such operative measures have been mentioned as are at present in use, while I considered it indispensable to include the important question of after-treatment.

Special care has been exercised in selecting the illustrations. The

wealth of illustrations of the anatomical section, including the diagrams, will no doubt contribute to a better understanding of the descriptive text. The majority of the illustrations have been made from my own preparations and cases, or drawn in accordance with my own clinical observations by the medical draughtsman, Carl Beck, in a very satisfactory manner. A number of illustrations referring to clinical findings have been supplied by Dr. Oskar Binesi; others, among which are the schematic sketches, have been drawn by myself.

Dr. Gustav Alexander.

Vienna.

Translator's Preface

No recent work has offered so much of value to the general practitioner and children's specialist as this by Professor Alexander of Vienna. The numerous illustrations assist in the thorough understanding of the many ear lesions and should make a strong appeal to the pediatrist who sees the conditions in daily work and to the otologist because of the exhaustive, authoritative statements relative to the common and more obscure conditions.

In this translation no additional notes have been added, the original text being complete, so that in presenting it to the English reading public a favorable reception is anticipated.

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Albany, N. Y.

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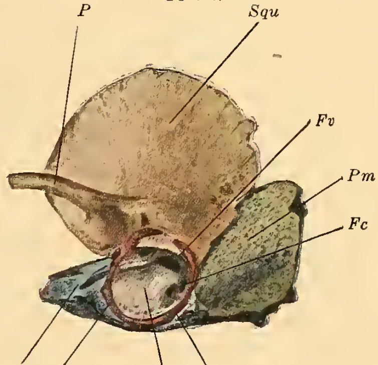
I. THE TEMPORAL BONE

THE bony foundation of the ear is formed by the temporal bone. In the newly born (Fig. 1) this consists of three sections, which in the course of further development grow together. They are the petrous, the squamous, and the tympanic portions. Posteriorly, the petrous portion ends in a broad base (mastoid part).

The squamous part of the temporal bone has two surfaces, an external and an internal. The upper edge of the squamous portion is connected with the parietal bone through the squamous suture, which admits only of slight dilatation of the skull. Anteriorly, the squamous bone sends forward the zygomatic process, which represents the border between the upper vertical and the lower horizontal portions of the squamous part of the temporal bone. The latter ends in two divergent cortical plates (Fig. 2); the upper one turns toward the anterior surface of the petrous bone and forms by junction with the similar petrous process the roof of the tympanum (paries tegminis, tegmen tympani) (Fig. 2) and the petrosquamous fissure. The lower cortical plate ends with a free edge, forming the lateral attic wall (Fig. 2, *la*). At the medial plane of the squamous portion there are the impressiones digitatæ, and a branching fossa destined to receive the middle meningeal artery. The anterior edge of the squamous portion forms with the petrous bone an interior angle in which the canal for the Eustachian tube and the tensotympani muscle terminates. Anteriorly to the auditory canal at the lower surface of the squamous portion there is the mandibular fossa, which is intended for the capitulum of the inferior maxilla. At the concave part of the tympanic ring (annulus tympanicus) there is the groove which will serve for the insertion of the tympanic membrane. The transformation of this ring into the tympanic bone is described on page 6.

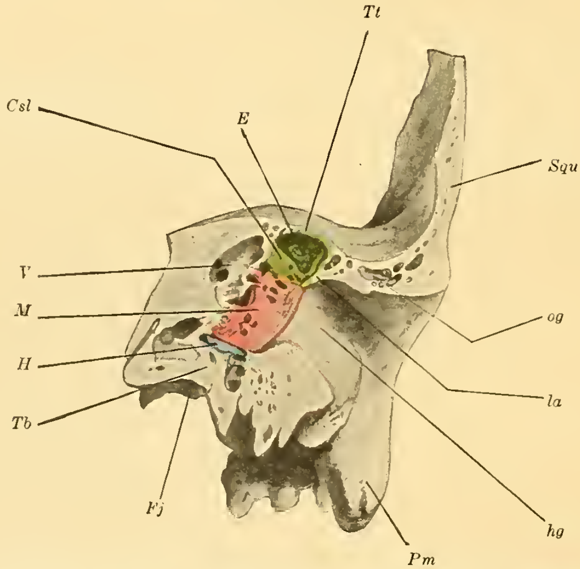
FIG. 1.



Temporal bone of the new-born, consisting of the squamous (squama temporalis, *Squ*), the mastoid (*Pm*), the petrous (*Pp*) and the temporal portions (annulus tympanicus, *At*), the latter of which becomes the tympanic portion. *Pz*, zygomatic process; *Fv*, fenestra vestibuli; *Fc*, fenestra cochleæ; *P*, promontory; *Fsm*, stylomastoid foramen.

If the squamous portion and tympanic ring are removed from the pyramid of the petrous bone in the new-born or in a preparation of the adult, a quadrangular pyramid will be seen, the base of which lies in the mastoid process, while the apex points forward and inward in the natural position of the skull. Two of the four surfaces are intracranial and two extracranial at the outer surface of the skull. The anterosuperior intracranial surface is called the cerebral surface, and the posteroinferior one the cerebellar surface. The upper edge of the petrous bone, which is bounded by the two surfaces, is also the border between the middle and posterior cranial fossæ. Posteriorly, the upper edge of the petrous bone

Fig. 2.



Vertical frontal section through the temporal cavity. Topographic arrangement of the tympanic cavity. *M*, mesotympanum; *E*, epitympanum; *H*, hypotympanum; *Pm*, mastoid process; *Fj*, jugular fossa; *Tb*, fundus of the tympanic cavity; *V*, vestibule of the labyrinth; *Csl*, protrusion of the horizontal semicircular canal; *Squ*, squamous portion of the temporal; *og*, superior auditory canal; *la*, lateral attic wall; *hg*, posterior auditory canal; *Tt*, tegmen tympani (paries tegminis).

runs into the upper edge of the transverse groove of the occipital bone. The cerebral surface of the petrous bone terminates exteriorly at the tegmen tympani and, in infants, is separated by the petrosquamous fissure from the corresponding squamous process, the upper osseous semicircular canal protruding ridge-like behind the middle of the plane. It forms the roof of the fossa subarcuata, a cavity the size of a small pea which becomes partly or completely obliterated at a later period. While this takes place, there will be osseous growth at the entrance to the fossa (Fig. 11), but the soft contents may remain intact for a long time, and remnants of it may even be preserved in adults. In some animals, especially the rodents, the fossa subarcuata is very roomy in spite of its relatively small entrance, and is filled with flocculus cerebelli.

In the new-born and infants during the first year of life, there is the superficial hiatus spurius at the lateral margin of the upper surface, which is a lateral aperture of the facial canal, the latter running at this place downward and backward. Later it is covered by a small plate of bone which is shifted over it from behind. The canal for the Vidian nerve extends from the hiatus to the apex of the petrous bone. At the apex itself there is the depression which is destined to receive the trigeminal ganglion.

The cerebellar surface of the petrous bone (Fig. 5) is bounded laterally and posteriorly by the sigmoid sulcus. At about the middle, the internal auditory meatus (meatus acusticus internus) is situated, with its longitudinal axis running a purely frontal course from right to left. Its length in the new-born does not exceed 4–6 mm., its full length of 10–12 mm. not being attained until puberty.

The fundus of the internal auditory canal is divided by a transverse crest into a small, superior, and a large, inferior, section. The nerves are inserted into their respective osseous canals within the fundus. The entrances are formed either by isolated gaps or cribriform apertures. One of the isolated gaps is intended for the facial nerve and another for the inferior ampullar nerve.

The osseous crest mentioned above and the area cribrosa which lies immediately underneath are intended for the accommodation of the nerves to the utricle and ampulla. The antero-inferior part of the fundus contains the tractus spiralis foraminosus, through which the cochlear nerve, coming from the cochlea, enters the internal meatus.

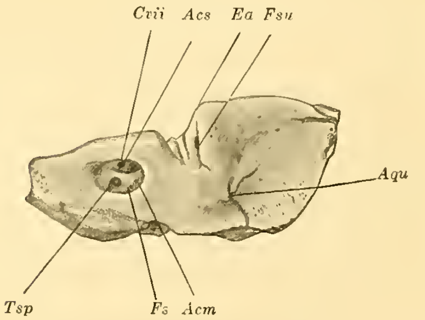
The arrangement of the cribriform gap of the tractus spiralis foraminosus indicates the spiral convolution of the cochlea. The area cribrosa media consists of a number of small gaps and is situated between the tractus and the superior macula cribrosa, receiving the nerve branches intended for the vestibular portion of the cochlear duct and saccule (Plate III, Fig. 3).

At about the middle, between the internal auditory meatus and the sigmoid sulcus, lies the external aperture of the vestibular aqueduct. In the first year of life it occupies a superficial position (Fig. 3); later on a small osseous plate grows over the aperture (apertura externa aquæductus vestibuli), similar to the osseous plate of the hiatus spurius facial canal, so that the aperture of the vestibular aqueduct assumes the shape of a longitudinal fissure and has the appearance of being laterally displaced (Fig. 4).

From above, the fossa subarcuata extends as far as the cerebellar surface of the petrous bone. After obliteration of this fossa there is sometimes a cicatricial cavity near the upper edge of the petrous bone, which may be mistaken for the external aperture of the aquæductus

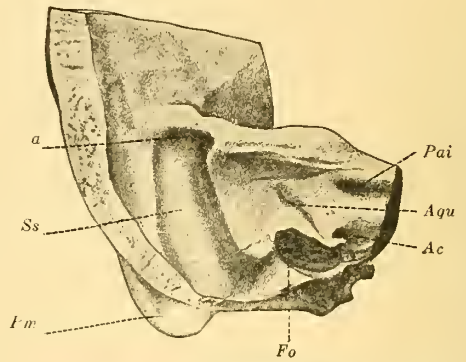
vestibuli. Below the aperture of the internal auditory canal, at the border angle of the cerebellar and inferior surface of the petrous bone, there is the triangular aperture of the cochlear aqueduct (Figs. 4–6).

Fig. 3.



Petrous portion of the tympanum in a six-months-old infant. Posterior surface of the petrous bone with the internal auditory meatus and the fossa subarcuata (*Fsu*). In the fundus of the internal auditory meatus is the aperture of the canal of the facial (Fallopii) (*Cvii*), area cribrosa superior (*Acs*), area cribrosa media (*Acm*), the foramen singulare (*Fs*), and the tractus spiralis foraminosus (*Tsp*). *Ea*, eminentia arcuata; *Aqu*, external aperture of the aquæductus vestibuli.

Fig. 4.



Posterior surface of the petrous bone in a ourteen-year-old child. Strongly developed angle (*a*) of the sigmoid sulcus (*Ss*). *Fo*, jugular foramen; *Pai*, internal auditory meatus; *Aqu*, aquæductus vestibuli; *Ac*, aquæductus cochleæ.

Fig. 5

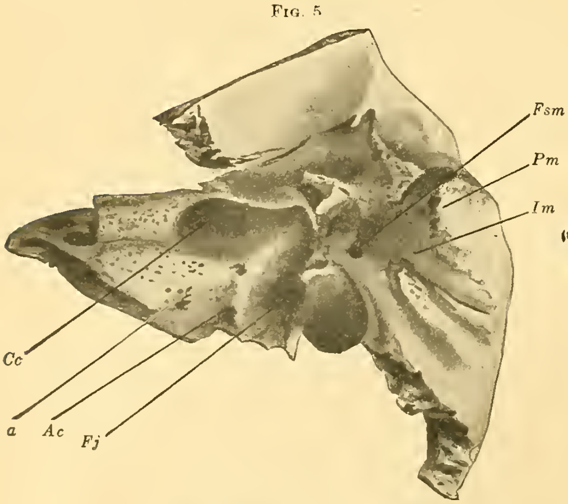
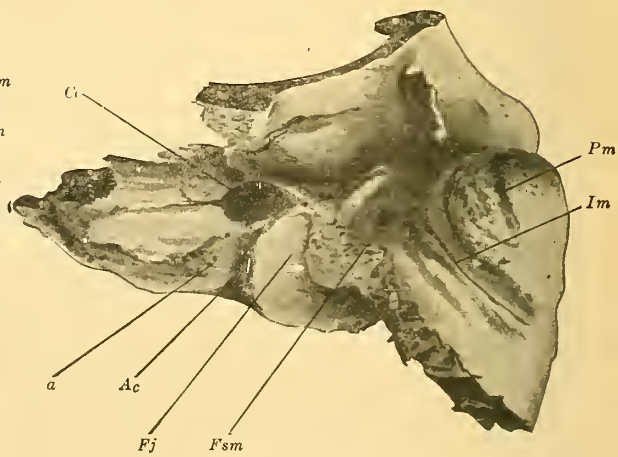


Fig 6.



Surgically important parts of the inferior surface of the temporal bone. *Cc*, carotid canal; *Ac*, aquæductus cochleæ; *a*, insertion field for the muscles of the tube and palate. Fig. 5. Deep and large jugular fossa (*Fj*); normal incisura mastoidea (*Im*). Fig. 6. Flat and small jugular fossa (*Fj*), apex of mastoid process (*Pm*). Incisura mastoidea only indicated (*Im*); it is so flat that the mastoid cells closely approach the stylomastoid foramen (*Fsm*).

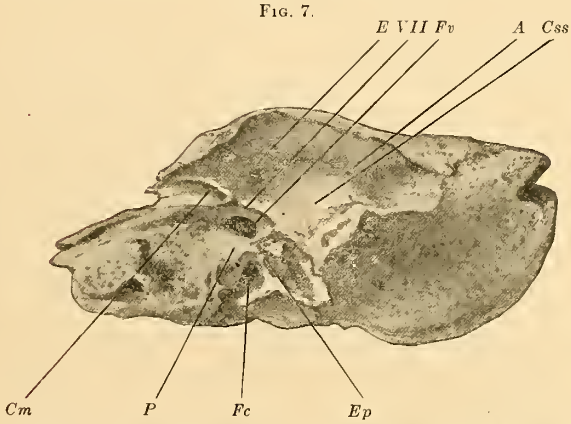
The medial part of the inferior surface of the petrous bone is occupied by the jugular fossa (Figs. 5 and 6). In front is the carotid canal, which traverses in semicircular form the anterior part of the petrous bone. There is also a rough area, the field of insertion for the Eusta-

chian tube and the soft palate. Behind the carotid canal is the flat fossula petrosa, from which a number of small canals lead to the middle ear.

Postero-exteriorly from the jugular fossa is the external aperture of the facial canal, the stylomastoid foramen; and from the end of the first year of life the styloid process can be found immediately in front of the facial canal and at the same depth.

The lateral surface of the petrous bone is only visible in the new-born and up to the end of the first year (Fig. 1); later on, it can only be seen after resection of the squamous part of the temporal bone and the tympanic bone. The central part of the surface is occupied by the vestibular window (fenestra vestibuli, Fig. 7), which is surrounded by the facial canal running in a postero-inferior direction toward the antrum. In the young, the facial canal is often detached close to the vestibular window, and the nerve at that place is only covered by connective tissue. This condition often exists to the fourth year, and explains the fact that in acute otitis media there is more frequently peripheral paralysis of the facial nerve in children than in adults.

Fig. 7.



Medial wall of the tympanic cavity of the new-born. *E*, epitympanum; *VII*, facial canal; *Fv*, fenestra vestibuli; *A*, antrum; *Css*, superior semicircular canal; *Ep*, eminentia pyramidalis; *Fc*, fenestra cochleæ; *P*, promontory; *Cm*, semicanalis muscle.

The lateral surface of the petrous bone has a groove-like cavity corresponding to the antrum; in the medial part of this cavity protrudes the external osseous semicircular canal in the shape of an oblong ridge. Behind the inferior pole of the vestibular window is the hollow pyramidal process, which is intended to receive the stapedius muscle. Before and below the vestibular window are the promontory and the fenestra cochleæ; above the promontory, runs more or less vertically the canal for Jacobson's nerve (Jacobsonii).

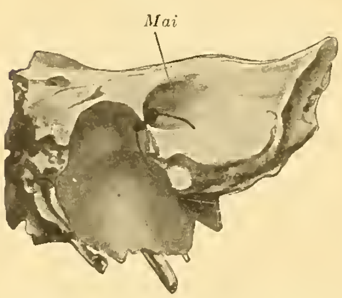
The tympanic groove extends upward to the hiatus spurius of the facial canal, while inferiorly it fuses with a short canal which terminates at the inferior surface of the petrous bone in the fossula petrosa. At the anterior pole of the vestibular window the processus cochleariformis is visible in the shape of a freely protruding osseous blade. This is the posterior end of the bony crest through which the canal for the Eustachian tube and tensor tympani muscle is divided into an upper and a

lower section. The crest running along the lateral surface of the petrous bone as a rule does not go beyond the middle of the lumen of the canal, but sometimes it extends further, and occasionally the osseous septum completely separates the canal into the two sections mentioned. The upper section is intended for the muscles of the tensor tympani, the lower one for the Eustachian tube (semicanalis tensoris tympani and seminalis tubæ).

II. CHANGES OF THE TEMPORAL BONE DURING GROWTH.

The temporal bone of the new-born consists of diploic bones, with the exception of the bony capsule of the labyrinth, the latter having at least a thin layer of compact bone. The upper ends of the tympanic ring blend with the squamous part in the first few months, the medial surface of the squamous portion growing almost simultaneously with the circumscribed places of the petrous bone (Fig. 10). The transformation of the tympanic ring into the tympanic bone is complete at the end of the fourth year, provided there is normal general development of the skeleton, but under certain circumstances the gap may persist longer and in rare cases even permanently (Fig. 9). Development of the mastoid process to any noteworthy size does not occur before the end of the first year. Obliteration of the fissura squamomastoidea commences at the end of the first year and is usually completed at the end of the second. Some sections of the petrosquamous fissure persist until the fourth year, sometimes permanently. The tympanosquamous fissure will permanently persist.

Fig. 8.



Posterior surface of the petrous bone. Jugular fossa enormously enlarged and extending to the vestibule and into the internal auditory canal (*Mai*).

The intramastoid fissure is seldom met with and, if present, obliterated at the end of the second year; its remnants, however, are sometimes demonstrable permanently in the shape of fossicular cavities.

The lateral surface of the petrous pyramid shows the smallest changes in the course of growth, showing merely a sharper outline and an increase in size corresponding to the growth of the entire pyramid. On the other hand, the jugular fossa at the inferior surface of the petrous bone grows considerably deeper, and the styloid process ossifies at the end of the first year. This may take place from the base (Politzer) or from the apex. Before both ossifications have reached each other, the ossified apex has become connected with the temporal bone by synchondrosis (Henle).

The following changes during growth of the cranial surfaces of the petrous pyramid should also be mentioned:

(1) Occlusion and obliteration of the fossa subarcuata;

(2) Covering of the superficial upper semicircular canal in the circular commissure and of the upper half of the sagittal semicircular canal by a compact osseous layer 2–5 mm. thick;

(3) Superposition of a compact layer, 3–7 mm. thick, over the upper pole of the cochlea;

(4) Growth of a protruding osseous layer over the hiatus spurius of the facial canal;

Fig. 9.

Persisting gap in ossification of the tympanum (*O*). *Mae*, external auditory meatus; *Pm*, mastoid process.

(5) Growth of a protruding osseous layer over the exterior aperture of the vestibular aqueduct;

(6) Prolongation of the cochlear aqueduct;

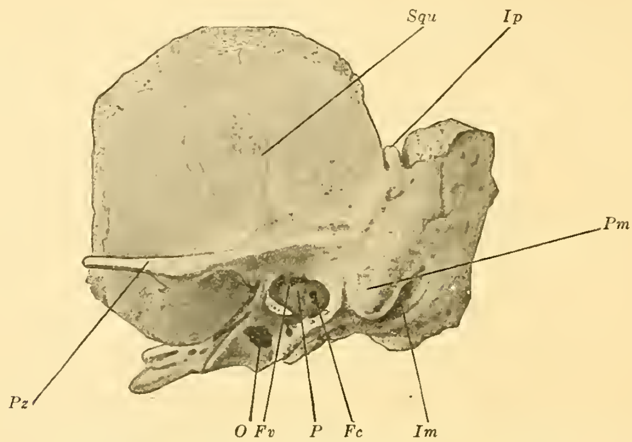
(7) Considerable elongation of the internal auditory canal with moderate narrowing of its superficial entrance;

(8) Deepening of the sigmoid sulcus; growth of the sulcus petrosus superior and inferior as well as of the sulcus petrosquamosus, provided a petrosquamous sinus has persisted.

Between the fifth and fifteenth years a gradual deepening of the groove for the middle meningeal artery and of the impressiones digitatae of the squamous part of the petrous bone occurs and may still further

increase in the course of later years. In exceptional cases the protrusion of bones may develop into commissures closing the sulcus petrosus or the sulcus arterial meningeal at circumscribed places in the shape of canals.

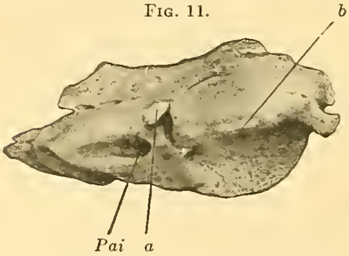
FIG. 10.



Lateral surface of temporal bone in a one-year-old child. *Squ*, squamous portion of temporal; *Im*, incisura mastoidea; *Fv*, fenestra vestibuli; *Ip*, incisura parietalis; *Fc*, fenestra cochleæ; *O*, gap of ossification; *Pm*, mastoid process; *P*, promontory; *Pz*, zygomatic process.

If the jugular fossa is very deep (Figs. 5 and 6), the fundus of the tympanic cavity becomes as thin as paper and may undergo cribriform perforation or complete detachment. In rare cases the jugular fossa may even extend to the internal auditory canal, so that the internal auditory canal will communicate with the jugular fossa by an aperture (Fig. 8).

FIG. 11.



Right petrous bone in a child seven months old. The fossa subarticulata is in the course of being obliterated by a growing protruding bone. *b*, superior edge of the petrous bone; *Pai*, internal auditory meatus.

There are three types to be differentiated in the topographical conditions from the sigmoid sinus to the mastoid process:

(1) The sinus, in normal position, runs in the projection of the posterior half of the mastoid process, while the anterior border of the sinus does not protrude beyond a straight line connecting the tuberculum supramastoideum with the apex of the mastoid process anteriorly.

(2) The second typical position is due to the sinus growing more remote from the mastoid process, the flat sigmoid fossa being shifted posteriorly and upward.

(3) The third typical position is caused by anteposition of the sinus and ectasis of the sigmoid sinus. In this case the anterior border of the sinus protrudes beyond the medial borderline anteriorly. In cases of extensive ectasis the tissue of the mastoid process is pushed to the area

of the apex, while the antrum region is occupied by the extended sinus. The latter is bluish-black in color and can be seen through the extremely thin lateral wall of the mastoid process.

III. THE EXTERNAL EAR.

The external ear consists of the concha and the external auditory meatus. The concha is formed of duplicature of skin which is kept in normal shape and position by the auricular cartilage. The concha in the child is remarkably soft and but slightly elastic in the first few weeks. At this stage its shape is rather shallow, while its retroverted margin (helix) is sometimes disproportionately large.

The growth of the concha during the first two years is remarkably rapid, the cartilage stiffens and its lateral surface deepens. During the same period the central parts (cymba conchæ, fossa navicularis, anti-helix) develop much more markedly than the marginal parts (Plate I, Fig. 1), as can be very plainly observed by comparing the length of the conchæ of two children, aged two and four, respectively (Plate I, Fig. 2). Pronounced growth of the free fold does not set in until the third or fourth year (Plate I, Fig. 3), while the lobe will usually not show any marked increase until puberty (Plate I, Fig. 4).

The external auditory meatus consists of a lateral and a medial section. The fundus of the lateral half is formed by connective tissue and cartilage (membrano-cartilaginous part), while that of the medial half is formed by bones (osseous part). The integument of the lateral part of the external meatus contains the normal papillæ, hairs, sebaceous glands and the ceruminal glands, the latter secreting a light yellow, clear, oily fluid. If this secretion is mixed with desquamated epithelial cells, dust, etc., a waxlike mass of yellowish-brown to black coloration will be formed in the outer meatus without any particular pathological cause (cerumen, ceruminal core).

The cartilaginous part can be stretched and distended. By traction it can be elongated by 4–5 mm., and by insertion of a small distending tube it may be widened by 3–2 mm. The integument of the osseous canal has neither hairs nor glands; it is attenuated toward the inner end of the canal, and the papillæ at that place become small and shallow. The subcutaneous cell tissue is but little developed at the end of the osseous canal in the vicinity of the tympanum.

The longitudinal axes of the cartilaginous and osseous canals join in an obtuse angle of 120–150°, which is open anteriorly and inferiorly. If the angle is very obtuse, the curvature of the entire external canal will, of course, be only slight and semicircular, while the angular kinking of the fundus of the external meatus at the transition of the membranous into the osseous part will be the more pronounced, the acuter

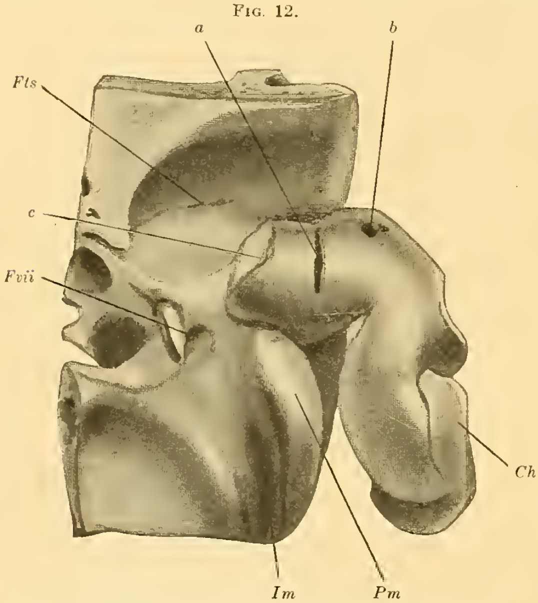
the angle. In the latter case, the "cellar" of the external canal is very deep, so that the antero-inferior quadrant of the tympanum is incomplete or quite invisible in the otoscopic picture.

There are three parts to be distinguished in the cartilage of the auditory canal: the tragus layer, the median commissure and the basal layer (Münch, Schwalbe).

By pulling the concha backward and upward, the auditory canal may be completely straightened out, especially in infants.

The movable and elastic properties of the membranous auditory canal are based upon the fact that the base of the cartilaginous canal is not formed by a cartilaginous tube, but a cartilaginous groove which is open backward and upward, while at its anterior and inferior walls it is provided with fissures. (Santorini's fissures.) Furthermore, the ear cartilage is not always directly inserted, but connective tissue may be interpolated at the lateral end of the osseous canal (Figs. 12 and 13).

Fig. 12.



Cartilage of the concha auriculi and external auditory meatus. Left ear, inferior aspect. Fissure (*a*) and gap formation (*b*) in the cartilage of the canal, which is inserted at the tympanum by a broad ligament of connective tissue. *Fts*, tympano-squamous fissure; *Fvii*, stylomastoid foramen; *Ch*, cauda helicis; *Pm*, mastoid process; *Im*, digastric fossa.

The greater part of the cross section of the external auditory canal is perfectly round. At the distal end of the membranous part, the cross section has sometimes a pronounced oval form with a vertical longitudinal axis, while the middle part of the osseous canal has in many cases the shape of a transverse oval. In other cases the border region at the posterior wall between the cartilaginous and osseous canals protrudes in a hump-like curvature, imparting to the cross section a bean- or kidney-like shape with a backward direction of the helix.

The anterior half of the concha is provided with sensitive fibres of the trigeminal, the posterior half with fibres of the great auricular nerve, coming from the superior cervical plexus, while the sensitive nerves of the exterior auditory meatus are supplied by the auricular branch of the vagus (Arnold's nerve). The latter enters the concha from below and behind, sending ramifications through the concha and

PLATE I.

Fig. 1. Normal auricle of the ear in a two-year-old boy. The central portions of the auricle, especially the conchial cavity, are relatively large. The lobule (*a*) is small.

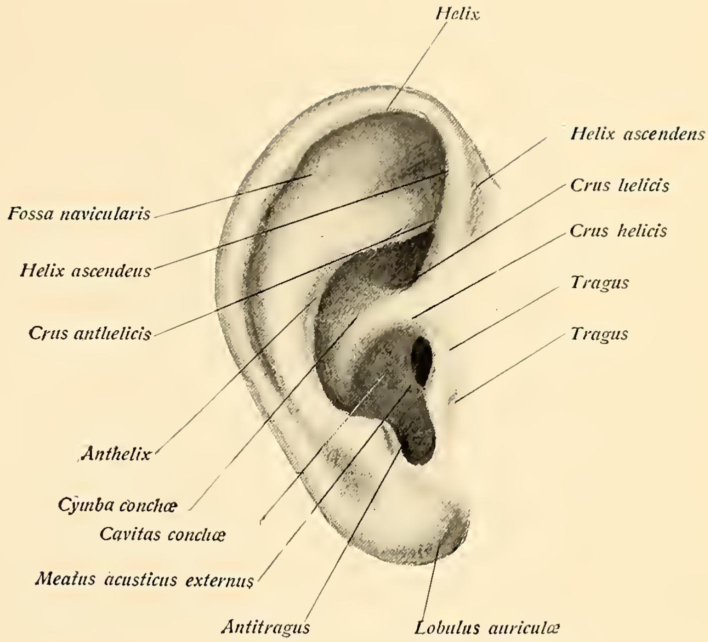


Fig. 2. Normal auricle of a two-year-old boy enlarged to the size of a four-year-old child. The disproportionate enlargement of the conchial cavity and the navicular fossa is seen by comparison with Fig. 3.

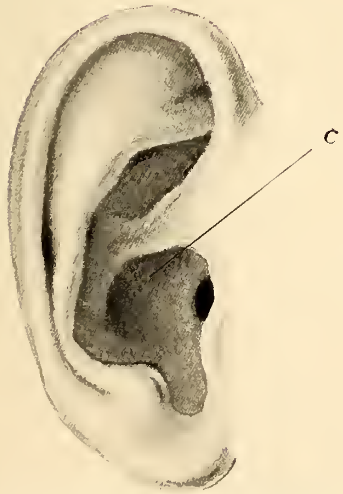


Fig. 3. Normal auricle of a four-year-old boy.

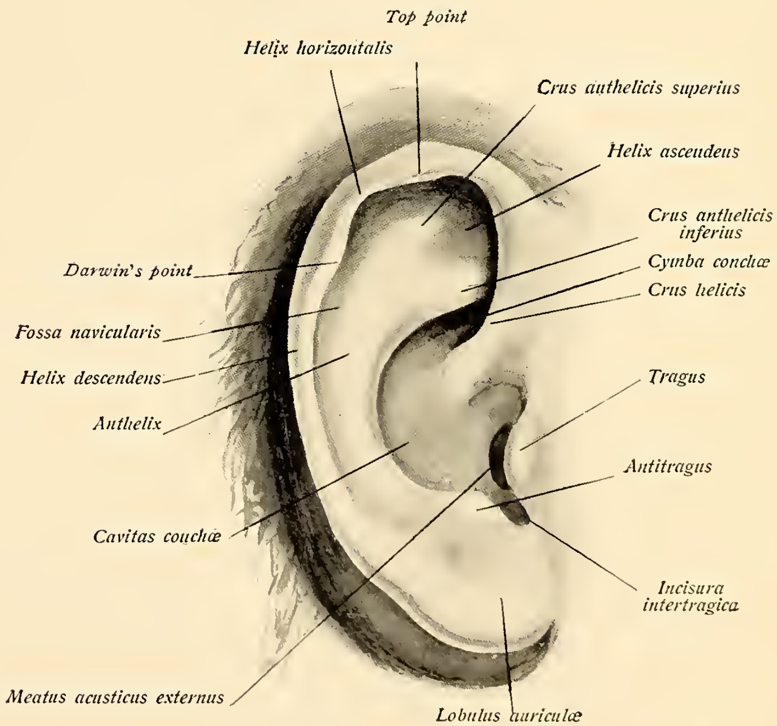


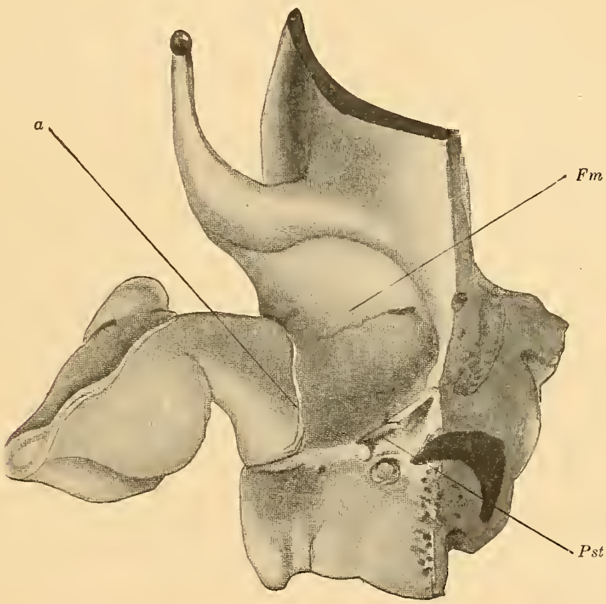
Fig. 4. Normal auricle of an adult.

into the posterior and inferior walls of the external canal. The motor nerves of the concha are supplied by the facial nerve.

The arteries of the external ear come from the area of the external carotid. The most important of these is the deep auricular artery which supplies the external auditory canal and leaves, together with the tympanic artery, the maxillary internal at the place where the latter crosses the neck of the capitulum of the submaxillary bone.

The postero-superior osseous wall of the auditory canal is formed by the anterior wall of the maxillary process and the lateral attic wall, while the exterior part is formed by the horizontal portion of the squa-

Fig. 13.



Direct communication between the cartilage of the auditory canal (*a*) and the tympanum. *Fm*, mandibular fossa; *Pst*, styloid process.

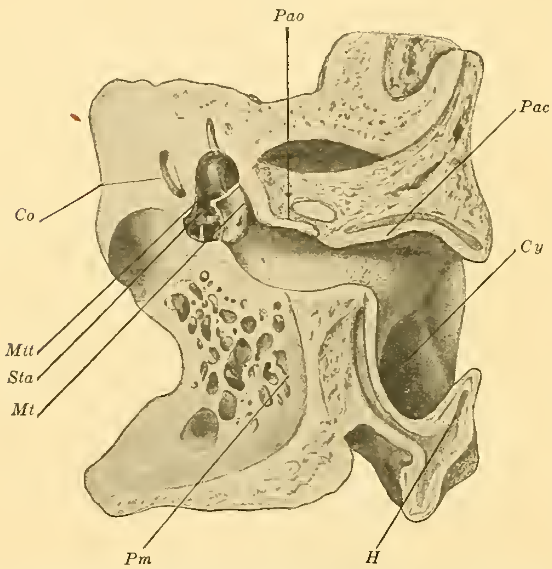
mous portion of the temporal bone. The antero-inferior wall of the osseous external auditory canal is formed by the os tympanum.

The development of the concha commences in the second embryonal month and starts from the sixth auricular eminence. These auricular tuberosities are grouped around the external auditory meatus. The external canal itself is developed rather late in the shape of a blind digitated process, commencing at the meatus. According to Urbantschitch, the occlusion of the external canal, both in the embryo and the new-born, is caused by embryonal agglutination, and the canal is seen to be completely filled with epidermal cells, both in the embryo and the new-born, sometimes even several weeks after birth.

The resorption of these cells commences after birth. The thick

epidermal layer becomes desquamated and is removed along with the secretion of the glands of the external canal. It is only rarely that the canal seems free from epidermal cells before birth, but even after complete desquamation of the embryonal tissue the auditory canal of the infant is not freely permeable, nor is it filled with air. Softness of the cartilage and absence of any osseous support cause the upper and lower walls to lie flat upon each other, so that the canal has the appearance of terminating in a fissure from an antero-superior to a postero-inferior direction. The osseous external canal is entirely absent in the new-born, as its surrounding osseous parts are not yet formed. The infant has no mastoid process, while the tympanic bone exists in the shape of a narrow, osseous groove in the shape of about three-quarters of a circle (Fig. 1), which is intended for the insertion of the tympanum and the membrano-cartilaginous canal. The membranous canal is connected with the almost horizontal lateral attic wall by thick connective tissue formation (Plate II). The permeability of the external canal becomes established at a later period when the cartilage becomes harder and more elastic. This rudimentary lumen is widened when the annulus tympanicus is transformed into the tympanic bone simultaneously with the glenoid fossa for the insertion of the capitulum of the submaxillary bone. The membrane which in the new-born occupies the place of the tympanic bone becomes gradually ossified.

Fig. 14.



Horizontal section through the left ear of a fourteen-year-old boy. Aspect of the superior half of the section. *Cy*, cymba conchæ; *H*, helix; *Pac*, anterior wall of the membrano-cartilaginous auditory canal; *Pao*, anterior wall of the osseous auditory canal; *Pm*, mastoid process; *Mt*, tympanic membrane; *Sta*, stapes with stapedius muscle; *Mtt*, tensor tympani muscle; *Co*, cochlea.

Ossification and increased growth of the newly-formed osseous layer take place from the margin, and this accounts for the fact that at the end of the second year there is a gap in the centre of the tympanic bone opposite the tympanum, which is occluded by connective tissue (Fig. 10). This gap disappears in many cases in the third year, but may persist longer, in exceptional cases even permanently (Fig. 9).

Formation of the posterior wall of the osseous external canal depends upon the development of the mastoid process, which is only present to any noteworthy extent after the infant is able to keep up and balance his head by the function of the cervical muscles. This, however, will not be the case before the end of the first year. In rachitis and arrested development the mastoid process is often rudimentary as late as the third and fourth years.

IV. THE MIDDLE EAR.

The middle ear is composed of the Eustachian tube, the tympanic cavity, the mastoid antrum and the mastoid process.

The tympanic cavity is divided into three spaces, according to the position they occupy toward the tympanum, as follows:

(1) Mesotympanum (median cavity of the tympanum, principal space of the tympanic cavity); (2) Epitympanum (cupola, attic or upper space of the tympanic cavity); (3) Hypotympanum (basal space, cellar of the tympanic cavity).

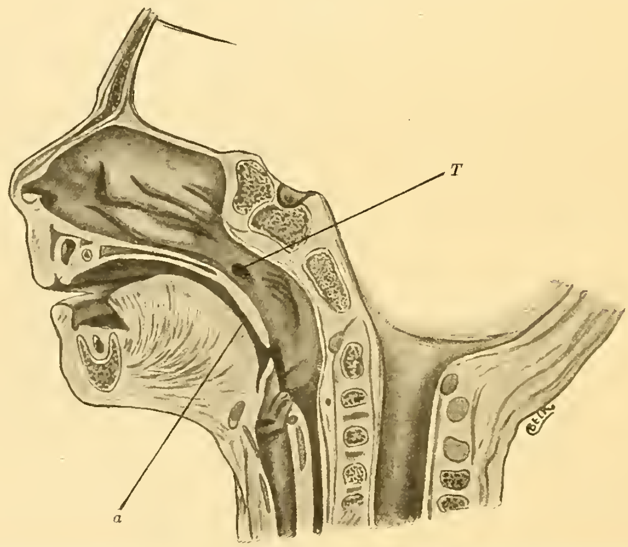
1. **The Eustachian Tube** (**tuba auditoria**).—The communicating tube between tympanic cavity and fauces consists of an osseous and membranocartilaginous part. The osseous part is situated in the lower portion of the canal or the Eustachian tube and tensor tympanic muscle and is bounded posteriorly and superiorly by the petrous bone. The tubal cartilage, which is bent in the shape of an S, is situated in the posterior wall of the cartilaginous tube (Fig. 16). It consists of spongy cartilage with occasional areolæ or fissures. The upper margin of the cartilaginous layer is bent laterally. The anterior wall of the cartilaginous tube is fibrous; the posterior cartilaginous wall is connected with the basal surface of the petrous bone by firm connective tissue, while the anterior wall is only united by loose connective tissue. The free margin of the cartilaginous layer bulges out in the shape of a ridge at the pharyngeal aperture of the tube (ostium pharyngeum tubæ), and this tubal ridge forms the anterior boundary of Rosenmüller's fossa. The latter is either present in rudimentary form in infants or entirely absent (Fig. 15). The tube crosses the pterygoid process in its course toward the pharynx. A few bundles of the musculus tensor veli palati are inserted at the lateral tubal wall, although some authors regard it as an entire muscle and designate it the dilator tubæ.

The cross section of the tube toward the pharyngeal ostium has a fissure in the shape of an S, and in a quiescent position the lateral wall lies closely against the medial one. As the cartilaginous tube approaches the osseous one, it becomes narrower, and its cross section is rounder; the narrowest part of the membranocartilaginous part (isthmus tubæ) is closed before its fusion with the osseous part. Having passed the

isthmus, the lumen gradually becomes larger again as it approaches the ostium tympanicum.

The osseous tube, together with the soft part immediately before it, is always open, while the rest of the membranocartilaginous tube is closed in the resting position. The longitudinal axis of the tube runs upward and outward, from behind, and the tube is generally perfectly straight. The cartilaginous tube contains many mucous glands (Fig. 16); its mucous membrane is covered with stratified, ciliated epithelium, extending upward to the ostium tympanicum, frequently even to the mucous membrane of the hypotympanum. It has numerous very fine longitudinal folds, while fissures in the tubal cartilage may lead to com-

Fig. 15.



Median sagittal section through the head of the new-born. *T*, position of the pharyngeal opening of the tube; *a*, velum of the palate.

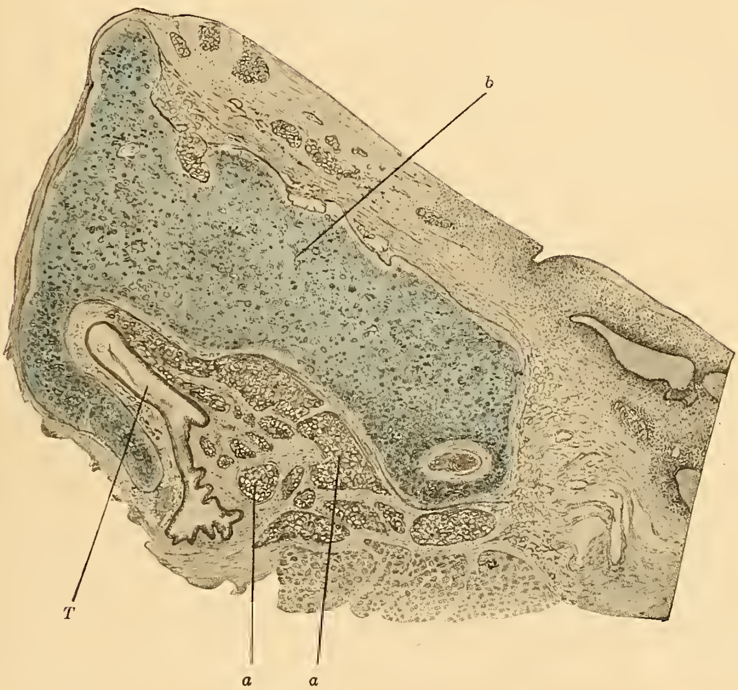
plete isolation of some cartilaginous parts. The connective tissue investing the surface of the tube, as well as the tympanic mucosa, is in direct communication with the connective tissue which fills the petrosquamous fissure in childhood. The entire tube in the new-born is 17–22 mm. long, relatively wide, and sometimes gaping in the cartilaginous part. The tubal cartilage is soft and not very elastic. It seems that the close approximation of the lateral and medial walls of the tube is only exceptionally present in the new-born and usually occurs in the first month. In the course of further development the pharyngeal opening of the tube gradually moves away from the choanæ at the lower margin, apparently proceeding upward toward the cranial base. This change of position depends on the development of the facial cranium in regard to size. The hard palate continues to gradually increase its

distance from the cranial base in the course of growth, whereas the tube which is affixed to the base of the skull remains topically unchanged in relation to the latter.

2. **The Tympanum.**—The tympanum is a quadrangular, prismatic space which is connected antero-inferiorly with the tube and postero-superiorly with the mastoid process by means of the antrum.

The longitudinal axis of the tympanum inclines considerably outward, forming an angle of 15–20° with the horizontal line in the new-born, and of 25–35° in the adult (inclination), while the tympanum

Fig. 16.



Cross section through the Eustachian tube. *T*, tubal lumen of the Eustachian tube; *b*, tubal cartilage; *a*, *a*, pituitary glands.

itself turns slightly forward and inward (declination), so that otoscopic clinical inspection from the exterior canal will make the postero-superior part of the tympanum appear much nearer to the eye than its antero-inferior section.

The lower wall (fundus) of the tympanic cavity is formed by a bony layer which originates at the petrous bone and, in the new-born, extends to the tympanic ring. In older children and adults this part is fused with the tympanic bone.

The lower surface of this osseous layer (Plate IV, Fig. 2) contains the fossa jugularis (Fig. 2), which is intended to receive the bulbus jug-

ularis. At its upper surface it is provided with narrow bony crests which, together with the entire osseous layer, represent the rudiments of the bulbus tympani. The latter is very strongly developed in some animals (equinæ, rodents, carnivora) and can still be demonstrated in the monkey. The thickness of the osseous layer varies considerably, being sometimes as thick as 5–8 mm., while in other cases it is as thin as paper and even sometimes punctured by small holes. In rarer cases it is entirely absent, so that the fundus of the tympanic cavity merely consists of connective tissue, and the bulb of the jugular vein protrudes into the tympanic cavity. If the fundus of the latter is very thick (Plate IV, Fig. 1), it consists of diploic bones or contains air cells (Fig. 2); if it is thin, it usually consists of a compact bony layer which does not contain very large areolæ.

The upper wall (paries tegminis) of the tympanic cavity (Plate IV, Fig. 3) is formed by the bony tegmen tympani which results from the union of a process emanating from the petrous bone with a corresponding plate of the horizontal portion of the squamous bone (Fig. 2). In the new-born, these bones are separated by the petrosquamous fissure, communicating only by connective tissue. The latter is in uninterrupted connection with both the tympanic mucosa and dura. The tegmen tympani does not exceed 1–2 mm. in thickness in the majority of cases; in others it is either as thin as paper, or has gaps or cribriform perforations. If the tegmen tympani is thick, the diploic tissue of the horizontal squama usually extends into the tegmen. The tympanic tegmen (paries tegminis) is the boundary between the tympanic cavity and the median cranial fossa. Anteriorly, it fuses with the cover plate of the musculo-tubal canal, posteriorly with the roof of the antrum.

The anterior wall of the tympanic cavity is formed by the osseous union between the anterior part of the tympanic bone and the petrous bone, with an aperture at the middle in the form of a canal. A narrow, bony crest, emanating from the petrous bone, divides the aperture and the attached canal into an upper and a lower section (Plate VII, Fig. 3).

The upper part of the canal is intended for the tensor tympani, the lower one for the osseous tube.

The posterior wall of the tympanic cavity consists of the anterior portion of the mastoid process with an aperture at the top the size of a small pea (aditus ad antrum), the cross section of which represents an equilateral triangle with the apex pointed downward (Fig. 2). Owing to the prominence of the lateral arch, the inner line of the triangle is slightly bulging; the upper line is formed by the roof of the antrum and the lateral one by the lateral wall of the attic and antrum.

The internal wall of the tympanic cavity is also called the labyrinthine wall. Its relief is very characteristic, and the centre is formed

by the vestibular window (Fig. 7). Before and under this window lies the promontory, and in the posterior margin of the latter there is the cochlear window, which is situated at the bottom of a small recess (fossula fenestrae cochleæ). There is a shallow furrow above the promontory (sulcus tympanicus s. Jacobsonii), at the end of which terminates the crista tympanica in a free outward eminence (processus cochleariformis). Posteriorly to the vestibular window, the facial canal bulges out (s. Fallopiae), and where the latter joins with the antrum, and in the antrum itself, there is the protrusion of the lateral semicircular canal. At the descending portion of the facial canal, immediately below the posterior margin of the vestibular window, there is the hollow pyramidal process, open at the apex, which is intended to receive the stapedius muscle, the tendon of which reaches the stapes through the aperture of the processus.

The lateral wall of the tympanic cavity is formed by bones in both its upper and lower sections (lateral attic wall, lateral wall of the hypotympanum). The aperture which remains at the middle is closed by the tympanic membrane (Fig. 22). The lateral attic wall represents the free terminating margin of the upper osseous wall of the auditory canal and is regarded as genetically contemporaneous with the squamous portion of the temporal bone (Fig. 2).

The other osseous parts of the lateral wall of the tympanic cavity connect with the tympanic bone, which is provided with a sulcus at the inner margin to receive the tympanic membrane. The aperture of the latter is round and has a small indentation at the antero-superior part (incisura Rivini), which arises from the fact that the curved tympanic bone is not closed at the antero-superior end. The lateral attic wall does not follow at this place the indicated circular line, but slightly recedes upward and outward. This causes the occurrence of small lateral osseous processes which are known as Helmholtz's processes.

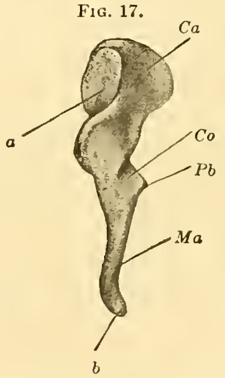
The lateral wall of the tympanic cavity is divided into three spaces for the requirements of the tympanic cavity: (1) The space lying in the projection of the tympanic membrane—the mesotympanum or principal space of the tympanic cavity; (2) the space immediately above, which is sometimes not much smaller than the mesotympanum, and is bounded by the lateral attic wall, and called the upper tympanic cavity, attic or epitympanum; it is of considerable clinical importance; (3) the space lying below the level of the tympanic membrane—the cellar or hypotympanum (Fig. 2).

The oblique position of the tympanic cavity is responsible for the fact that, in the otoscopic picture, nearly the entire upper half of the tympanic wall up to the posterior part of the vestibular window appears to belong to the epitympanum. The lower half of the medial wall and the major portion of the base of the tympanic cavity belong topograph-

ically to the mesotympanum. The hypotympanum, therefore, appears as a shallow triangular groove.

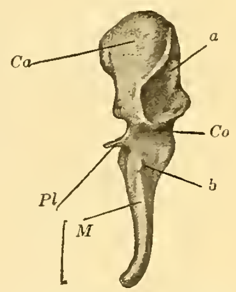
The lateral and medial walls of the tympanic cavity are connected by the chain of auditory ossicles (Figs. 17–21). The system of this chain in the human being can be directly derived from the simple conditions prevailing in reptiles and birds, where a bony rod runs transversely through the middle ear, with its external end affixed to the tympanic membrane and its medial part to the vestibular window. This columnar rod is called "columella." In mammals, as well as in the human being, there is a chain of three ossicles instead of one columella. The lateral end of the columella, which is connected with the tympanic membrane, corresponds to the malleus in man (Figs. 17, 18 and 20).

Fig. 17.



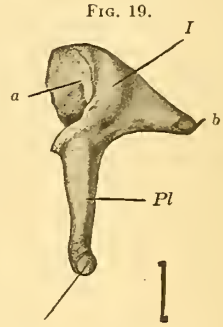
Right malleus, lateral aspect. (3.5 : 1.) *a*, articular surface for connection with incus; *Ca*, head (capitulum) of malleus; *Co*, neck (collum) of malleus; *Pb*, short process (P. brevis); *Ma*, manubrium; *b*, grooved end of manubrium.

Fig. 18.



Right malleus, internal aspect. (3.5 : 1.) *Ca*, head of malleus; *a*, articular surface for connection with incus; *Co*, neck of malleus; *b*, place of insertion for musc. tensor tympani; *M*, manubrium of malleus; *Pl*, processus longus.

Fig. 19.

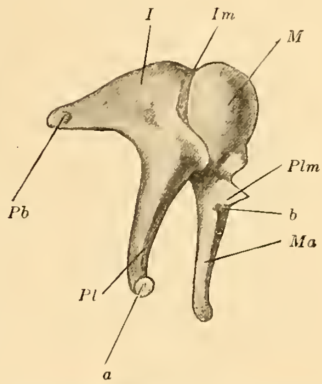


Right incus, interior aspect. (3.5 : 1.) *I*, body of incus; *a*, articular surface for connection with malleus; *b*, place of insertion for posterior malleolar ligament; *Pl*, processus longus; *c*, process of stapes.

The malleus consists of the head, neck and manubrium. Head and neck form with the manubrium an obtuse angle of about 140°. At the place of juncture between manubrium and neck there is a protruding, button-shaped, short process, while anteriorly a slender, nail-like long process runs toward the tympanosquamous fissure. In the new-born the long process shows a leaf-like broadening and is almost as long as the entire malleus. The peripheral part of the long process undergoes rapid involution, appearing in a six-months infant almost shorter than the short process, as in the adult. At the medial point of juncture between manubrium and neck there is a punctiform, rough spot where the tendon of the tensor tympani muscle is inserted. At the posterior circumference of the head there is a saddle-shaped, articular surface for the incus.

The exterior form of the incus (Figs. 19 and 20) resembles a tooth with two roots and a poorly developed crown. The medial part is called the body of the incus, and has at its medial plane a shallow fossa occluded by connective tissue. A cone-shaped, horizontal process with posterior aspect is called the short process; the long process, which runs vertically downward, is flexed at a right angle at the end. The terminal part is distended in the shape of a small button and carries the articular surface to connect with the incus and the head of the stapes. The flexed, button-shaped end of the incus stands, so far as history of development is concerned, in relation to the long crus, as the epiphysis does to the diaphysis, although normally their osseous union occurs *in utero*. Should this osseous union fail to take place, the lower end of the long crus of the incus may appear as an independent part in macerated preparations. In former times it was designated "ossiculum Sylvii."

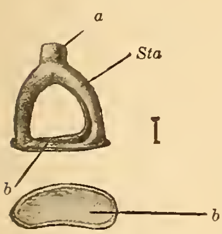
Fig 20.



Right malleus and incus in natural position, interior aspect. (3.5 : 1.) *I*, incus; *Im*, syndesmosis between malleus and incus; *M*, malleus; *Plm*, long process of malleus; *b*, place of insertion for musc. tensor tympani; *Ma*, manubrium of malleus; *a*, articular surface of incus for the anterior stapes articulation; *Pl*, long process of incus; *Pb*, short process of incus.

The posterior end of the short crus of the incus has a flattened surface like a facet. The body of the incus has a gable-shaped articular surface corresponding to the impression of the articular surface of the malleus. The lateral surface is provided with small processes pointing backward and downward, regulating the movements of the malleus and incus articulation in such a way that any inward movement of the malleus takes the incus along with it, while the latter may remain in a partly or wholly stationary position in any outward movement of the malleus.

Fig. 21.



Right stapes. (3.5 : 1.) *a*, head of stapes; *b*, plate of stapes; *Sta*, crura of stapes.

The stapes (Fig. 21) consists of the plate, the crura and the head. The plate is kidney-shaped, the hilus pointing downward and forward. The crura form hollow grooves of extreme tenderness. The tendon of the stapes muscle is inserted in the posterior crus near the head.

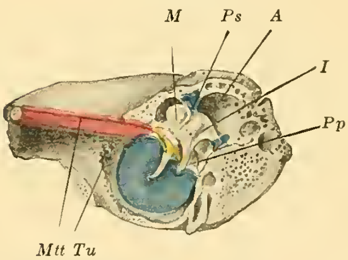
The malleus in the new-born is 7.5–8.5 mm. long; the long crus of the incus 6.5, the short one 4.25 mm.; the height of the stapes is 3.25 and the length of the plate 2.5 mm. After birth the auditory ossicles increase by about one-tenth their original size, but the ossicles vary in size and shape, the stapes undergoing the greatest deviations, especially in regard to position and length of crura, less so the incus, and least of all the malleus.

Both malleus and incus are derived from the first postoral arch, and originate from the hindmost section of Meckel's cartilage, from the anterior part of which the submaxillary bone is developed as a membrane-bone. Stapes and pedicular process as well as the stylo-hyoid ligament and the inferior epihyal bone, are descendants of the second postoral arch.

The connection between malleus and incus as well as between incus and stapes is usually a simple syndesmosis, positive articulations with demonstrable rudimentary articular cavities and accessory ligaments having only been found in rare cases. In many animals with an excellent sense of hearing (rodents and others) the articular surfaces of malleus and incus are joined by osseous growths.

The stapes is fixed in the vestibular window by an annular ligament which imparts to the stapes considerable motility, especially after the union between the incus and stapes has been severed. The manubrium of the malleus is completely embedded in the tympanic membrane (Figs. 23–25).

Fig. 22.

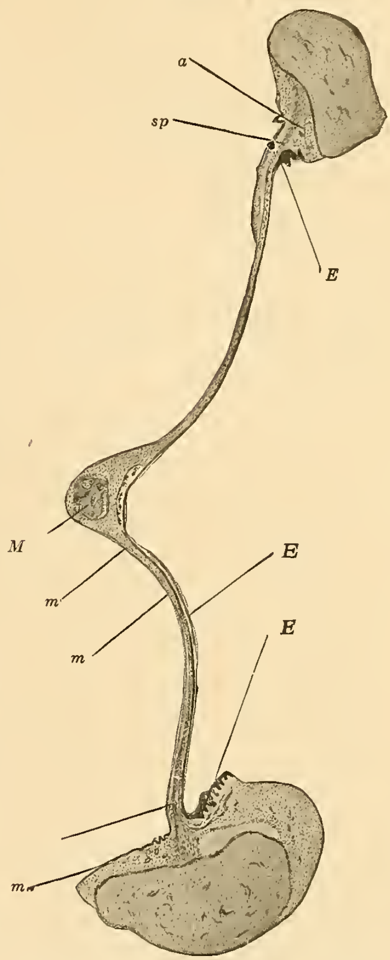


Right tympanic membrane and auditory ossicles of a two-months-old infant. *M*, malleus; *I*, incus; *Ps*, fold between malleus and incus (sup. ligament of mall.); *A*, antrum; *Pp*, posterior incus fold; *Mtt*, musculus tensor tympani; *Tu*, tuba auditiva.

The tympanic membrane is divided into two parts: a large one—pars tensa—and a small one—pars flaccida (membrana Shrapnelli). The former is intimately adherent to the manubrium up to the short process and spread out in funnel-shape from this adhesion (Fig. 23). It has a diameter of 7.5–9 mm. in the new-born and of 9–11 mm. in the adult. The central and lowest point of the funnel coincides with the lowest point of the manubrium (umbo). The tympanum has three layers; the lowest, which consists of connective tissue (lamina propria), shows a combination of radial and circular fibres (stratum radiatum, stratum circulare). The radial fibres are particularly well developed in the middle of the tympanic membrane and along the manubrium, and occasionally form a yellowish white spot at the umbo demarkated by rays (pes anserinus). The circular fibres are situated medially from the radial fibres and are particularly frequent at the peripheral margin of the tympanic membrane (Fig. 23), where they occasionally form a grayish-white ring, which is demonstrable as early as at the end of the first year, but is more frequently observed at the clinical examination of old individuals with atrophic integument of the auditory canal. The manubrium is surrounded by circular fibres and layers of connective tissue which terminate in looplike figurations (Fig. 25). The circular connective tissue layers

which surround the manubrium are of very firm texture and give the impression of bones at the otoscopic examination. In this way the manubrium appears much thicker in the picture of the normal tympanic membrane than would correspond to the actual circumference of the bony structure (Fig. 24). In a serous, osmotic tympanic membrane, however (suppurative catarrh of the middle ear), the connective tissue layers become transparent, and the outline of the manubrium now appears narrow and thin in the otoscopic picture (Plate VIII, Fig. 1).

Fig. 23.



Horizontal section through the tympanic membrane of a new-born child at the level of the manubrium (*M*). *E*, epidermis of the external auditory meatus; *sp*, substantia propria; *m*, mucosa layer of tympanic membrane; *a*, accumulation of circular fibres at the peripheral end of tympanic membrane.

The substantia propria of the tympanic membrane is invested with a layer of epidermic epithelium toward the auditory meatus, this layer being considerably thicker in the first few months of life than in the adult. During the first six months there is rather considerable desquamation, and if in the first few weeks there is rapid desquamation of the newly-developed epidermal surface, there may appear small epidermal nodules which microscopically are seen to contain cholesterine crystals and may simulate cholestoma. No papillæ can be recognized in the epidermic layer of the pars tensa.

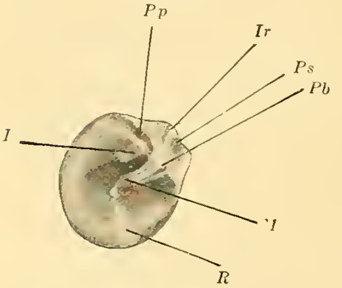
The medial plane of the substantia propria is invested with the tender epithelium of the tympanic mucosa, the greatest part of which is cuboidal, while in the basal part of the tympanic membrane and in the direction toward the tubal ostium it is cylindric and in the upper part of the membrane frequently consists of pavement epithelium. The mucous layer of the membrane, in conjunction with the lamina propria, forms folds and trabecular thickening which, in a flat aspect, appear as a dendritically ramified network (Gruber) (Fig. 29).

The pars flaccida (membrana Shrapnelli), which varies in size from a pinhead to a hempseed, has only one layer of the substantia propria (Fig. 27), and the arrangement in circular and radial fibres is likewise absent (Fig. 27). There are papillæ in the epidermal layer, and the

layer of mucosa adheres in the first year, and sometimes longer, to the ligaments and folds of the mucosa of the upper space of the tympanic cavity.

The stapes muscle (musculus stapedius) is situated in the pyramidal process, which allows the muscle tendon to reach the stapes through its upper end. The malleus muscle, or tensor tympani, originates in the upper section of the musculo-tubal canal. Its tendon embraces the processus cochleariformis, traverses the tympanic cavity, and thus reaches the malleus (Figs. 30 and 31). The stapedius muscle is innervated by a branch of the facial nerve, the malleus muscle by a branch from the trigeminal, one coming from the interior pterygoid nerve and the other from the otic ganglion.

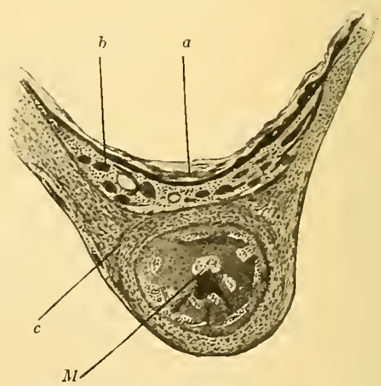
Fig. 24.



Picture of normal tympanic membrane. *M*, malleus; *Pb*, processus brevis; *Ps*, plica superior; *Pp*, plica posterior; *I*, incus; *R*, light spot; *Ir*, incisura Rivini.

The chain of auricular ossicles is supplied with a number of ligaments, one of which encircles in convolutions the manubrium immediately below the short process. It originates at the medial surface of the lateral attic wall. The manubrium continues its course antero-inferiorly to the tympanosquamous fissure. The supero-posterior part of this ligament is described as the external malleus ligament (ligamentum mallei externum) and the antero-inferior part as the internal malleus ligament (ligamentum mallei anterius) (Fig. 27).

Fig. 25.



Cross section through the tympanic membrane of a new-born at the level of the manubrium. *M*, manubrium; *c*, circular connective tissue layers encircling the manubrium; *b*, vessels of the manubrium; *a*, epidermal layer, abundant desquamation of epithelium.

The incus ligament originates in two portions at the external wall of the antrum and is inserted at the posterior end of the crus of the incus (Fig. 32). The auricular ossicles, as well as their muscles and ligaments, are invested with mucous membranes forming folds which at some places are of regular appearance, and at others variously shaped (Figs. 27 and 28).

The typical folds of the mucous membrane (Figs. 22, 30, 31 and 32) are the following:

(1) The malleus-incus fold, wrongly designated as malleus-incus ligament or as upper malleus ligament. It runs from the upper pole of the malleus-incus articulation to the roof of the tympanic cavity;

PLATE II.

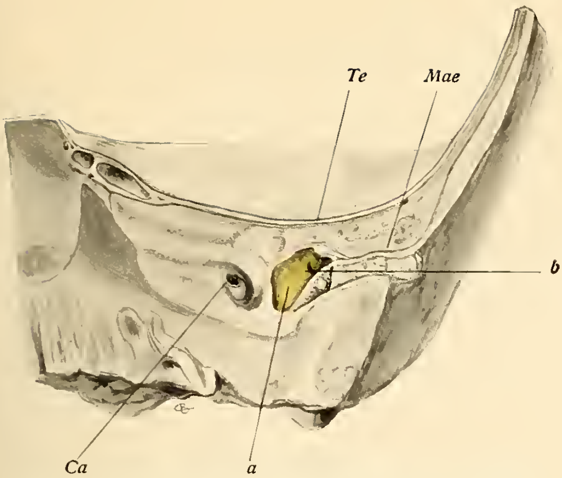


FIG. 1. Frontal vertical section through the ear of the new-born. Mucous tissue (a) in the anterior part of the tympanic cavity near where the tube branches off. Enlarged 1.5 : 1. b tympanic membrane; Te, tegmen tympani; Mae, external auditory meatus; Ca, internal carotid.

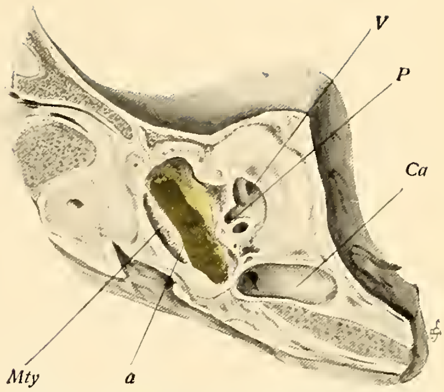


FIG. 2. Vertical section through the ear of the new-born. In the area of the promontory (P) and the vestibule (V), the chain of auricular ossicles is invested with embryonal mucous tissue (yellow, a), which completely fills the entire tympanic cavity. Enlarged 1.5 : 1. Mty, tympanic membrane; Ca, internal carotid.

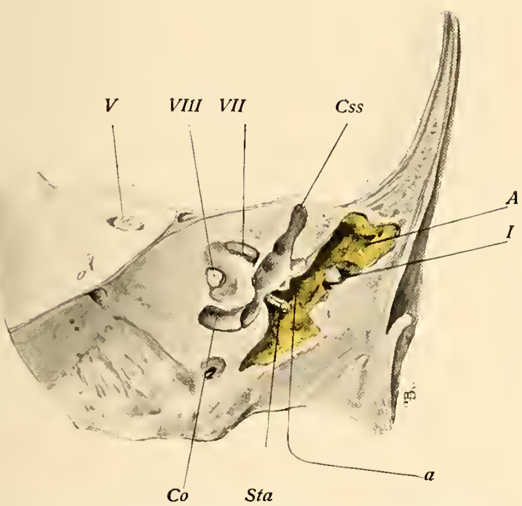


FIG. 3. Frontal vertical section through the ear of the new-born in the area of the antrum (A). Posterior half of section. Enlarged 1.5 : 1. The entire space of the middle ear is filled with mucous tissue (a), from which the short incus process (I) and the stapes (Sta) protrude. A, attic; Css, superior semicircular canal; Co, cochlea; V, trigeminal nerve; VII, facial nerve; $VIII$, auditory nerve.

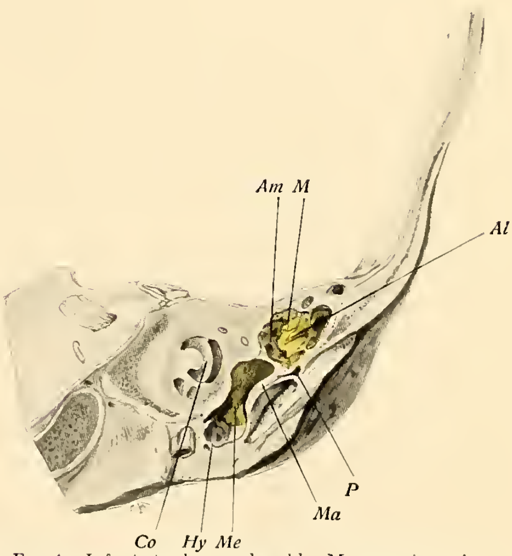


FIG. 4. Infant, twelve weeks old. Mucous tissue in the epitympanum. Head of malleus (M) completely covered with mucous tissue. The lateral (Al) and the medial (Am) attic are almost completely filled with mucous tissue. The meso- and hypotympanum are already free and passable. P, Prussak's space; Ma, manubrium; Me, mesotympanum; Hy, hypotympanum; Co, cochlea.

(2) The anterior malleus fold. It encloses the anterior malleus ligament, the chorda tympani and the long process of the malleus;

(3) The posterior malleus fold between the manubrium and the posterior edge of the tympanic membrane;

(4) The fold of the chorda;

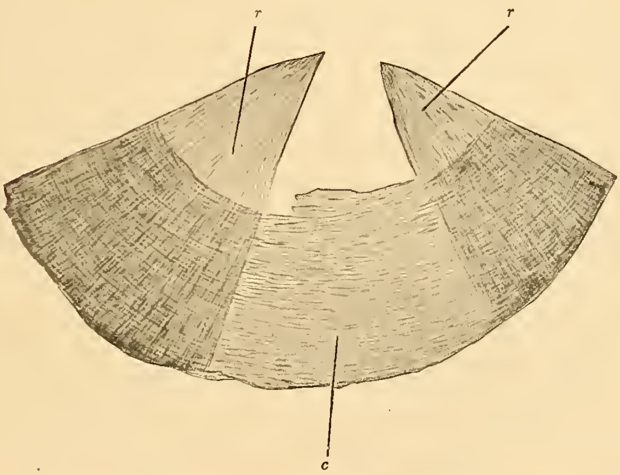
(5) The stapes fold. It partially or completely occludes the lumen bounded by the crura and plate of the stapes.

The following belong to the atypical folds:

(1) A mucomembranous bridge between the tympanic bone and the tensor tendon;

(2) A mucomembranous bridge between the manubrium and the long incus process;

Fig. 26



Radial (*r*) and circular (*c* fibres of the tympanic membrane. Preparation in glycerin.

(3) Several mucomembranous folds extending from the body of the incus to the lateral and medial walls of the tympanic cavity;

(4) Slender radial mucomembranous bridges, running from the stapes to the edge of the fossula fenestræ vestibuli (Politzer). Besides, there are small spherical or oblong corpuscles which sometimes occur in the tympanic cavity and more frequently in the antrum (Politzer's corpuscles).

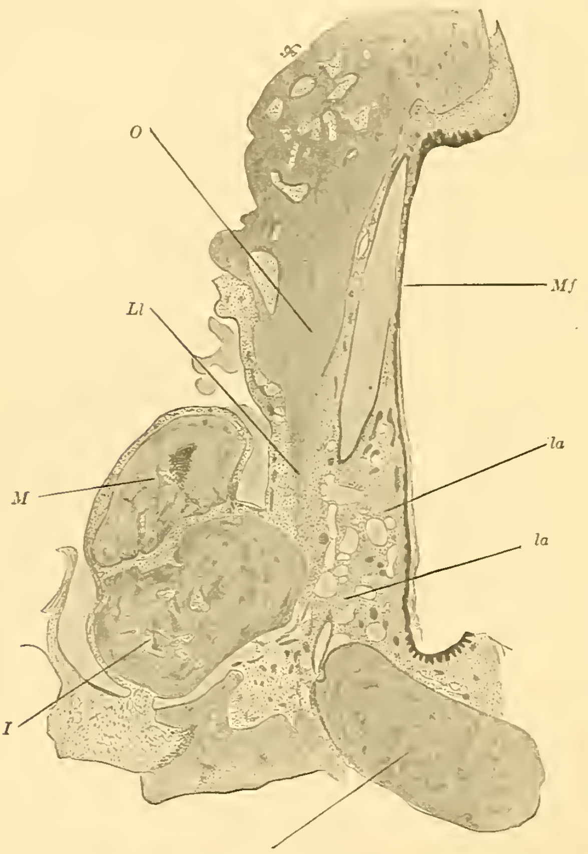
All the folds of the mucous membrane are normally transparent like glass, tender and elastic in the living as well as in the fresh preparation. In the latter they often become visible at the moment of tearing the preparation with a needle. Through catarrhal and other affections of the middle ear the folds may become gray, thickened and rigid (chronic adhesive process).

The folds of the mucous membrane represent the remnants of an extensive mesodermal tissue (Plate II), which fills the entire tympanic

cavity of the fœtus as well as the new-born (Hyrtl, v. Troeltsch). The tissues becomes resorbed, as soon as air enters through the Eustachian tube, while they will undergo rapid purulent disintegration from inflammation of the middle ear in the new-born or a few weeks after birth.

After death the mucous tissue undergoes exceedingly rapid decomposition from putrefaction, filling the tympanic cavity of infants with a gelatinous, more or less purulent fluid.

Fig. 27.



Horizontal section through the tympanic membrane of the new-born at the level of Shrapnell's membrane (*Mf*) and the malleus-incus articulation (*M*, *I*). The lateral attic (*la*) is filled with embryonal mucous tissue. *O O*, annulus tympanicus at the level of the incisura Rivini; *Ll*, lateral ligament of malleus; *Mf*, membrana flaccida Shrapnelli.

In the normal course of development, resorption of the mucous tissue occurs first in the hypotympanum, while the mucous membrane of the tympanic cavity remains permanently thicker in the hypotympanum than in any other area of the middle ear, abundant folds and lymphadenoid tissue being not infrequently found in children four to five years old. It has even been proposed to designate the latter as tympanic tonsils. The lymphatic tissue is preserved a little longer in the folds of the mucous membrane in the shape of small-celled infiltrates (Anton). Resorption of the mucosa deposits in the mesotympanum is usually complete in the sixth to eighth week. The mucous membrane of the tympanic cavity becomes thin, but the folds permanently persist. The mucous tissue of the tympanic cavity is preserved longest in the upper space, which, in most cases, is still traversed by large numbers of reticularly ramified mucous bridges. These are sometimes demonstrable as late as the first and, in arrested development and under-nutrition, the second year.

The tympanic cavity is supplied with blood from three arterial areas:

(1) Internal maxillary artery, which sends a branch, the tympanic artery, into the tympanic cavity by way of the tympano-squamous fissure;

(2) Branchlets of the internal carotid and the posterior auricular. The stylomastoid artery, which fuses with the chorda tympani and is a small branch of the ascending pharyngeal artery, reaches the tympanic cavity through the tympanic canal;

(3) Several branches of the middle meningeal artery.

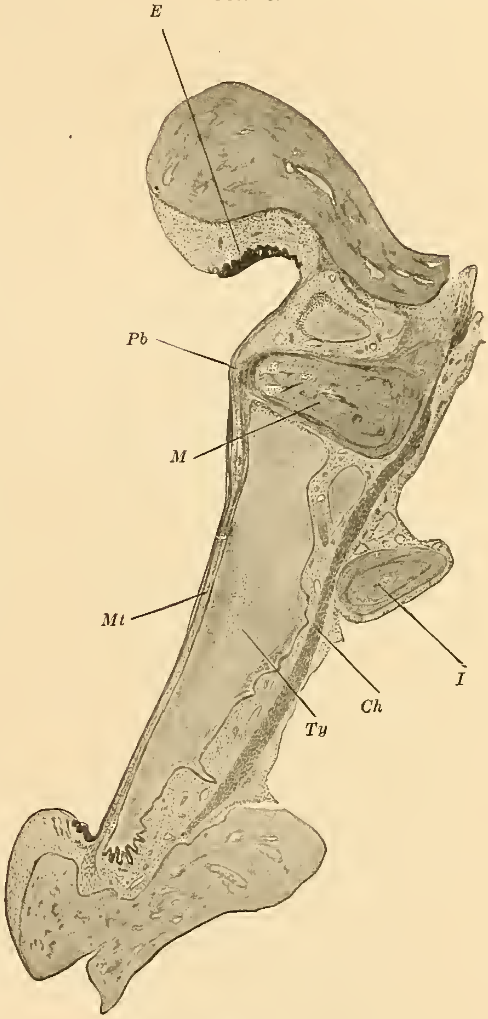
All these vessels form a very dense vascular net, anastomose with each other, and sometimes communicate either direct (Fig. 45) or through the artery of the internal auditory meatus with the vessels of the labyrinth (Politzer).

There are two vascular areas in the tympanic cavity:

(1) A network of capillaries near the posterior margin of the manubrium, which fuses with the blood vessels of the external meatus at the upper pole of the tympanic membrane;

(2) Circular capillaries at the peripheral margin of the tympanic membrane, which exclusively emanate from the blood vessels of the middle ear.

Fig. 28.



Horizontal section through the tympanic cavity (*Ty*) of a new-born at the level of the short process (*Pb*) and the chorda tympani (*Ch*). *M*, malleus; *I*, incus; *Mt*, membrana tympani; *E*, epidermis of the external auditory canal.

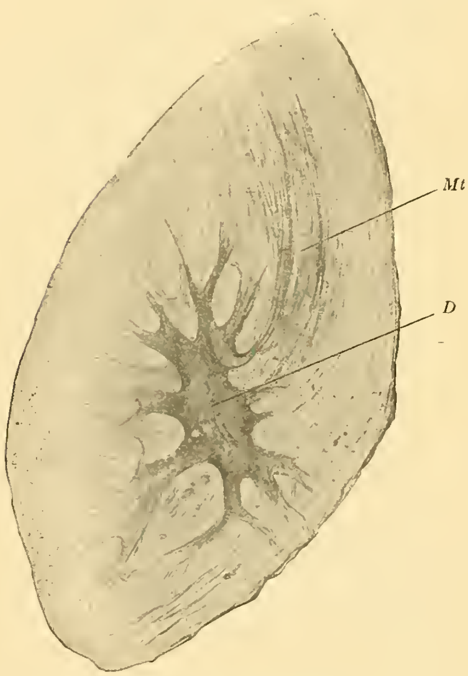
The vessels of the manubrium are connected by a dense radial network with peripheral circular vessels.

The lymph vessels of the middle ear are supplied by the area of the deep cervical lymph glands, the bundle of submaxillary glands and the retropharyngeal glands. On the other hand, the lymph vessels of the

middle ear do not communicate with the mastoid glands on the planum mastoideum which contains the lymph vessels of the occipital portion of the scalp.

The tympanic cavity is supplied with sensitive fibres by the tympanic nerve (Jacobson's), which originates in the area of the glossopharyngeus and, ascending from the otic ganglion, reaches the tympanic cavity, proceeds along the promontory upward to the geniculate ganglion and, fusing in the tympanic cavity with the sympathetic fibres of the carotid plexus, finally dissolves in the plexus tympanicus. Secretory and sensitive fibres are left behind in the geniculate ganglion, where they join the descending facial and leave it again as chorda tympani (Figs. 31 and 32). The latter takes an arcuate course through the tympanic cavity, arrives between malleus and incus anteriorly at the tympanosquamous fissure, through which it leaves the tympanic cavity without giving off any branches.

Fig. 29.



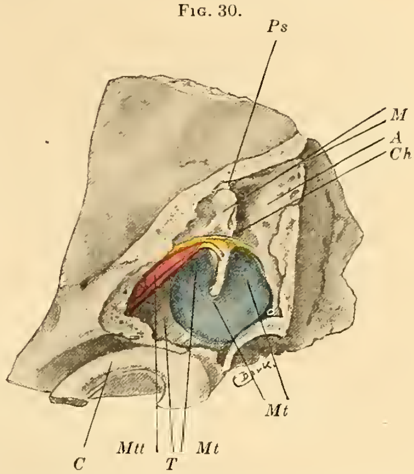
Dendritic network (*D*) of the medial surface of the tympanic membrane (*Mt*).

3. The Topography of the Middle Ear.—The ligaments and mucous folds of the auricular ossicles and tympanic cavity are for the most part situated in the border region of the meso- and epitympanum, causing slight perforated or fissured connections, even in normal conditions. The base, or lowest point of the epitympanum, in the natural position of the head, is formed by Shrapnell's membrane. The communication between the meso- and epitympanum may be entirely destroyed if the normal folds of the mucous membrane become enlarged and thickened through catarrhal inflammations. Should this lead to suppurative inflammation of the upper tympanic space, the pus will be unable to escape into the mesotympanum and gradually gravitate toward Shrapnell's membrane, which will eventually be perforated. Isolated perforation of that membrane is, therefore, always a sign of suppuration of the middle ear confined to the upper space of the tympanic cavity. Insertion of malleus and incus materially reduces the free space of the attic, and there occurs a cleft between the lateral attic wall and the auricular ossicles; and a

similar cleft between the latter and the medial tympanic wall. The first cleft is called the lateral, and the second the medial attic.

The portion of the upper space between the tympanic cavity lying in the projection of the membrana flaccida is called Prussak's space (Plate II, Fig. 4). It is bounded exteriorly by Shrapnell's membrane and the free border of the osseous attic wall, superiorly by the lateral ligament of the malleus, interiorly or medially by the neck of the malleus, inferiorly by the short process of the malleus. Prussak's space opens anteriorly and posteriorly into the attic.

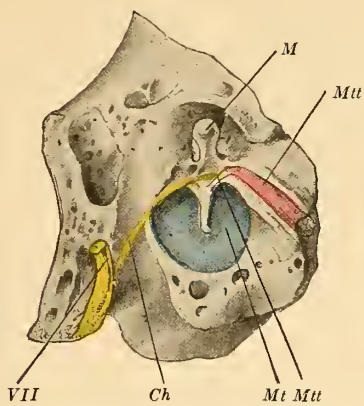
FIG. 30.



Right lateral wall of the tympanic cavity, interior aspect. *Ch*, chorda tympani; *Mt*, tympanic membrane; *Ps*, superior malleus fold; *M*, malleus; *A*, tympanic antrum; *C*, carotid; *T*, tuba auditiva; *Mtt*, tensor tympani muscle.

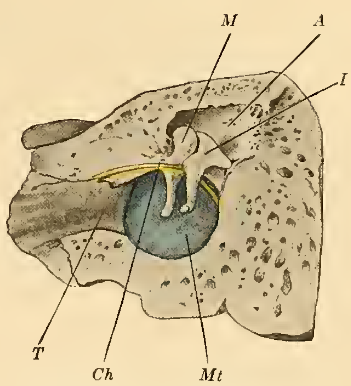
The clefts bounded by the malleus folds and the tympanum are called tympanic pockets (Figs. 30, 31 and 32). The chorda pocket lies between the posterior tympanic fold and the chorda fold. The anterior tympanic pocket and the chorda pocket are closed at the top, while the posterior tympanic pocket communicates at the top with Prussak's space.

FIG. 31.



Left lateral wall of the tympanic cavity, interior aspect. Course of the chorda tympani. ($1\frac{1}{4}$ of natural size.) *VII*, facial nerve; *Ch*, chorda tympani; *Mt*, membrana tympani; *Mtt*, tensor tympani muscle; *M*, malleus.

FIG. 32.



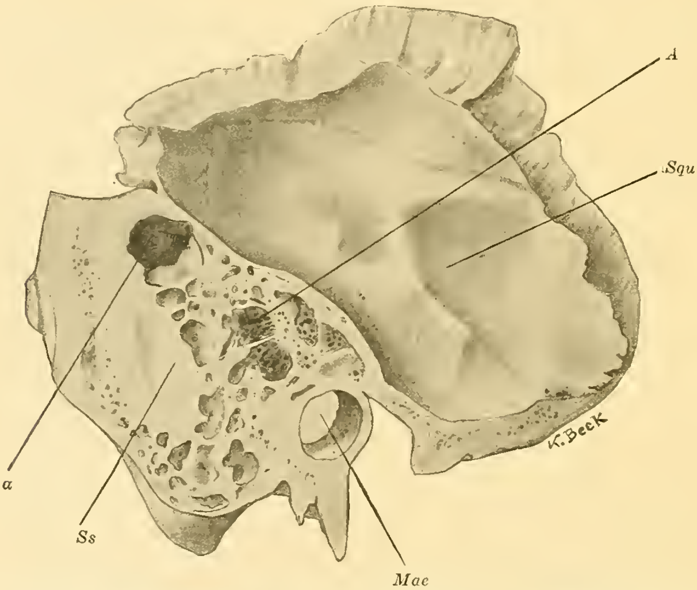
Right lateral wall of tympanic cavity, interior aspect. ($1\frac{1}{4}$ of natural size.) *Ch*, chorda tympani; *T*, tuba auditiva; *M*, malleus; *I*, incus; *A*, tympanic antrum; *Mt*, membrana tympani.

This connective aperture, however, is frequently obliterated by catarrhal affections, but owing to its small size, even when present, it plays no material part in the downward drainage of the upper tympanic space.

4. **The Tympanic Antrum.**—The antrum is a cavity the size of a small pea, which is inserted between the tympanic cavity and the mastoid process. Its upper wall is formed by the tegmen antri, the lateral wall by the free border of the superior and posterior auditory canals (anterior wall of the mastoid). At the lateral wall there is the fossa incudis, to which the posterior incus ligament adheres.

The osseous lateral semicircular canal protrudes at the medial plane in the shape of an oblong crest. Posteriorly, the antrum terminates in the shape of a spherical cavity with cribriform perforation of the wall. The gaps correspond to the ostia of the mastoid cavities which, in normal

Fig. 33.



Flat section through antrum tympanicum (*A*) and the mastoid process. *Ss*, sigmoid fossa; *a*, upper pole cell; *Mae*, external auditory meatus; *Squ*, squamous portion of the temporal.

cases, communicate with the antrum. The latter is invested with a tender mucous membrane and traversed by a few thin mucous folds which frequently contain Politzer's corpuscles. It can at once be demonstrated with a normal preparation that a colored fluid, introduced into the antrum, penetrates into all the mastoid cells. The antrum is already developed in the new-born and situated superficially, owing to the complete absence of the mastoid. The medial part of the lateral wall at birth, and sometimes a few months later, contains cartilaginous remnants. Up to the end of the second year the osseous walls remain extremely thin, and this explains why in juvenile cases of mastoiditis subperiosteal abscesses and osseous fistulæ develop very rapidly in the mastoid region.

5. The Mastoid Process.—The mastoid region of the new-born is flat, the process itself is absent; antrum, digastric fossa and the stylo-mastoid foramen are superficially situated. The mastoid process develops with the increase in size of the sternocleidomastoid muscle and is primarily to be regarded as a muscle process, serving to enlarge the fold of insertion for the muscle. The development of the mastoid, therefore, occurs simultaneously with the rapid increase in thickness of the sterno-cleidomastoid, which takes place as soon as the infant tries to carry or balance its head independently. A really important development of the mastoid, however, will not commence before the end of the first year, when the infant commences to walk and attempts to exercise its balancing power, so that the gradual development of the mastoid is

Fig. 34

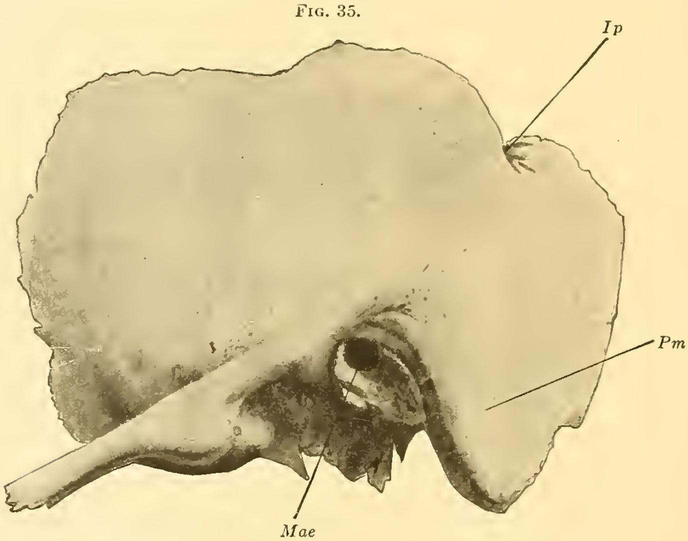
Lateral surface of temporal bone of twelve-year-old child.

intimately connected with the general physical development. Any disease arresting the ability of controlling movements of the head and attempts at walking will interfere with the development of the mastoid. At the age of one year the structure of the latter corresponds to that of the other cranial bones. The outer layer is rather thin, and its interior is entirely filled with diploic tissue. At the age of two years the normal development of the air cells commences. The growth of the mastoid is practically completed at puberty, but there will still be increase in thickness of the outer layer and a further extension of the cells in the vicinity of the sinus and apex.

At the lateral surface of the mastoid there are the small mastoid fossæ, which deepen toward the auditory meatus, the mastoid triangle and the antrum triangle. At the border of the external auditory meatus,

there is the suprameatal spina. The mastoid is bounded by the crista temporalis inferior toward the squama. At the normal position of the head the crista is about at the level of the base of the medial cranial fossa; its posterior end is broadened and elevated in the shape of a flat eminence (tuberculum supramastoideum). The upper and medial parts of the lateral mastoid surface are smooth except for an ethmoidal punctation near the mastoid fossa. Short periosteal layers of connective tissue run into the small mastoid fossæ. The rough apex of the mastoid is the field of insertion for the sternocleidomastoid. At the medial plane of the mastoid there is the digastric fossa, which carries at its medial border the pedicular process and the lower aperture of the facial canal (foramen stylomastoideum).

Fig. 35.



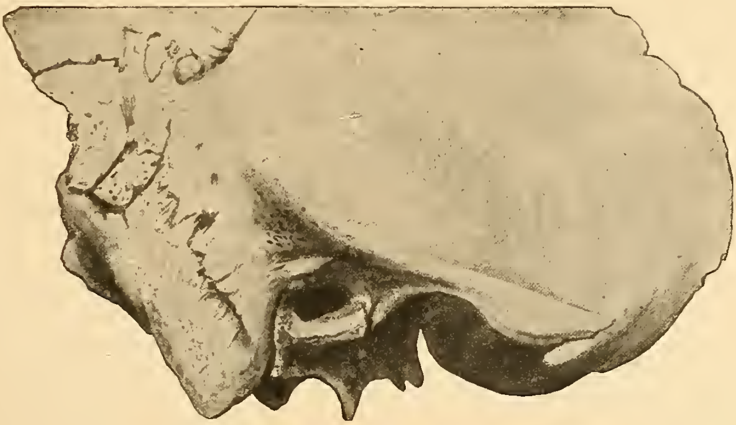
Temporal bone of fourteen-year-old boy with congenital stenosis of the exterior meatus (*Mae*). *Ip*, incisura parietalis; *Pm*, mastoid process.

The border between the mastoid and squama in the new-born is the squamomastoid fissure which divides the mastoid into a small antero-posterior and a large postero-inferior part, and occasionally persists either completely or partly until the end of the second year in the shape of a fissure directed towards the base of the auditory meatus. On the other hand, the intramastoid fissure runs from the incisura parietalis to the mastoid apex, and is a variety of the first-named fissure.

The fissures are filled with connective tissue and have plenty of blood-vessels which serve for the better nutrition of the rapidly growing bony parts. Remnants of these fissures are not infrequently demonstrable in old age in the shape of clefts or indentations filled with partitions of the periosteum and sometimes conduct mastoid veins outside. In rarer cases, the vascular canals will persist after obliteration of the

fissure, arranged in a vertical line in accordance with the direction of the fissure. Occasionally any one of the fissures may persist completely (Fig. 36).

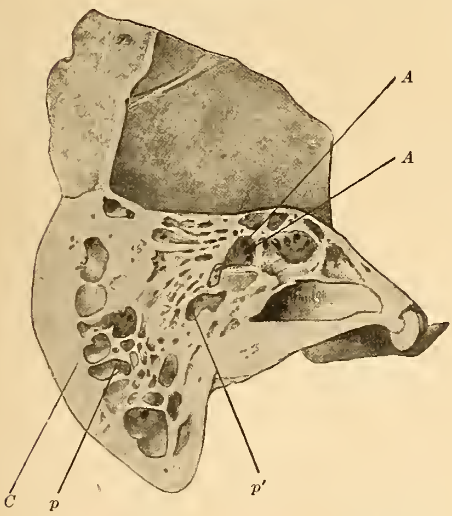
FIG. 36.



Temporal bone with persistent squamomastoid fissure.

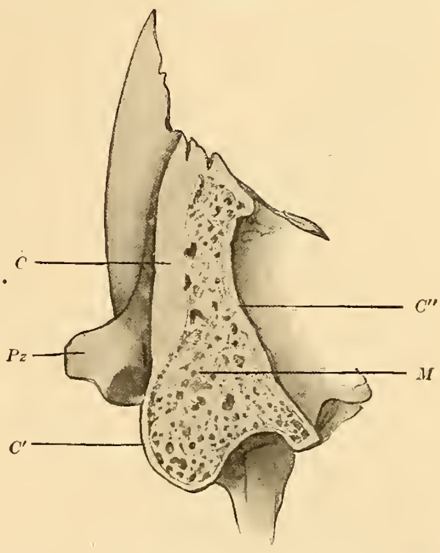
The surface layer of the fully developed mastoid is thickest at its lateral surface and becomes rapidly thinner toward the apex. At that

FIG. 37.



Vertical section through a pneumatic mastoid (*p*). *p'*, antrum cells; *C*, exterior (thick) layer; *A*, antrum tympani.

FIG. 38.



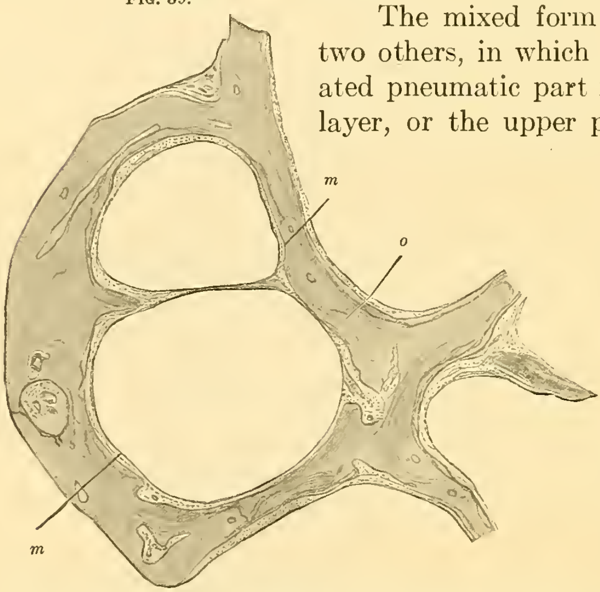
Vertical section through a diploic mastoid (*M*). The external corticalis (*C*) is thin at the apex (*C'*); the inner corticalis (*C''*) is likewise thin. *Pz*, zygomatic process.

place, and at the medial wall, where it is often as thin as paper, it fuses with the compact portion of the petrous bone (Fig. 38).

The mastoid process contains air-carrying, pneumatic cells or diploë

and, accordingly, a distinction is made between the pneumatic, the diploic and the mixed mastoid. The pneumatic mastoid (Fig. 37) may be large- or small-celled, and it is only exceptionally that it is made up of a few large cavities. The pneumatic cells are invested with a tender, pale mucosa and communicate with each other by apertures. Occlusion of some or all of the communicating apertures, and therefore partial isolation or obliteration of mastoid cells, may occur from inflammatory affections. Large cells occur at the apex and near the sigmoid sinus (Fig. 33) of the pneumatic mastoid, while in the vicinity of the antrum the cells are usually small (Fig. 37). The diploic mastoid contains plenty of blood and lymph spaces (Fig. 38). The mixed form is a combination of the two others, in which either the centrally situated pneumatic part is invested with a diploic layer, or the upper portion of the mastoid is pneumatic, the apex diploic, or the reverse. In the juvenile mastoid, which is always diploic, the development of pneumatic spaces means a secondary change in which the diploic tissue is displaced by the pneumatic and finally resorbed. If this displacement and resorption is complete, the consequence will be a purely pneumatic mastoid; if some diploic parts remain, the consequence is a mixed mastoid. If no areolas are formed at all, the mastoid will be diploic even in the adult. In advanced age, the osseous tissue of the mastoid may be either exceedingly thin (osteoporosis) or very dense (osteosclerosis). These changes are merely incidental to advancing age and do not signify any aural affection. In cases of chronic suppuration of the middle ear, however, a more or less extensive condensation of the bony structure in the interior of the mastoid will occur, amounting to pathological osteosclerosis of the mastoid. In some of these cases the bone turns as hard as ivory and completely loses all its cavities (eburnation of the mastoid).

Fig. 39.



Normal pneumatic tissue of the mastoid. *o*, bony trabecula; *m*, mucous membrane.

According to size and form, we have to distinguish between (1) the normal mastoid; (2) the small mastoid; (3) the large, bullous mastoid; (4) the flat mastoid; and (5) the pointed mastoid.

PLATE III.

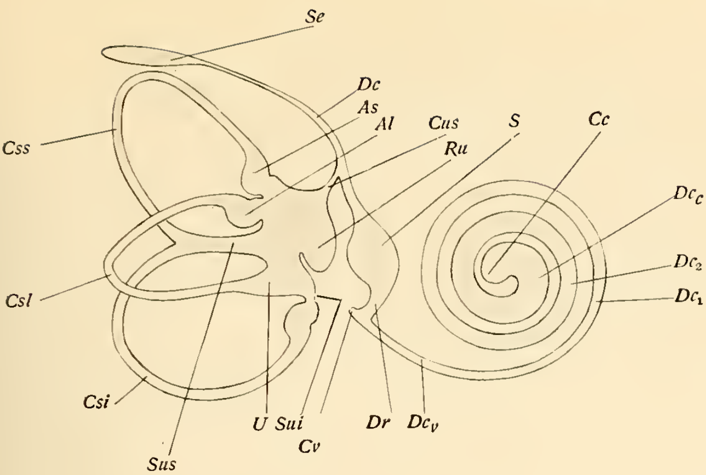


Fig. 1. Diagram of membranous labyrinth. Superior portion: *Css*, superior semicircular canal; *Csl*, lateral semicircular canal; *Csi*, inferior semicircular canal; *As*, superior ampulla; *Al*, lateral ampulla; *Ai*, inferior ampulla; *Sus*, superior utricular sinus; *Ru*, anterior utricular sinus (recessus utriculi); *Sui*, inferior utricular sinus; *U*, utricle. Inferior portion: *S*, saccule; *Cus*, utriculo-saccular canal; *Dc*, ductus endolymphaticus; *Se*, saccus endolymphaticus; *Dr*, Ductus reuniens; *Cv*, vestibular cul-de-sac of cochlea; *Cc*, cupolar cul-de-sac of cochlea; *Dcv*, vestibular section of cochlea; Dc_1, basal convolution of cochlea; Dc_2, medial convolution of cochlea; *Dcc*, apical convolution of cochlea.

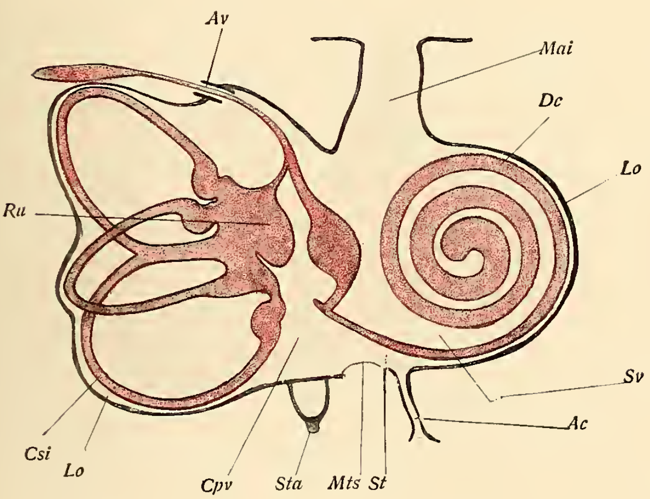


Fig. 2. Topography of osseous capsule of labyrinth (*Lo*) and of the membranous labyrinth (diagram). *Av*, vestibular aqueduct; *Mai*, internal auditory meatus; *Ac*, cochlear aqueduct; *St*, scala tympani; *Mts*, membrana tympani secundaria; *Sta*, stapes; *Ru*, recess of utricle; *Csi*, inferior semicircular canal; *Cpv*, cisterna perilymphatica vest.; *Sv*, scala vestibuli; *Dc*, cochlear duct.

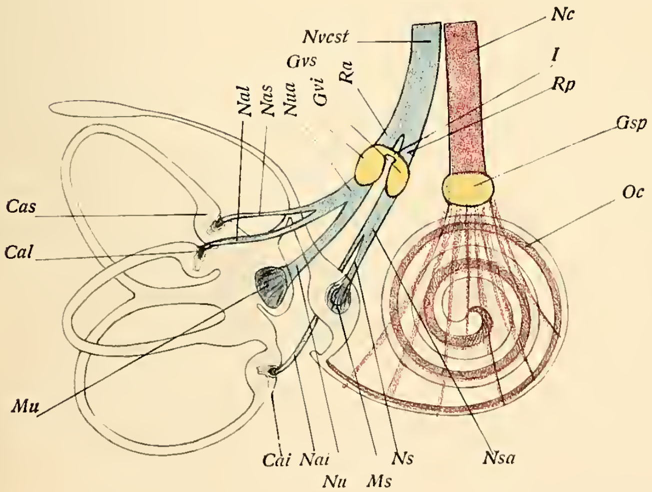


Fig. 3. Ramification of nerves of the membranous labyrinth and its nerve-endings (diagram). *Nvest*, vestibular nerve; *Nc*, cochlear nerve; *Gvs*, superior vestibular ganglion; *Gvi*, inferior vestibular ganglion; *Gsp*, spiral ganglion; *Nua*, utriculo-ampullar nerve; *Nu*, nervus utriculi; *Nas*, superior ampullar nerve; *Nal*, lateral ampullar nerve; *Nsa*, nervus sacculo-ampullaris; *Ns*, nervus saccularis; *Nai*, nervus ampullaris inf.; *Cas*, crista ampullaris sup.; *Cal*, crista ampullaris lat.; *Cai*, crista ampullaris inf.; *Mu*, maculi utriculi; *Ms*, maculi sacculi; *Oc*, organ of Corti.

The small mastoid is mostly diploic and is found more frequently in the female sex and individuals with weak musculature. The bulbous mastoid is always pneumatic. Expansion of the areolæ downward

Fig. 40.

Normal temporal bone with conical mastoid process; oval cross section (12-year-old child).

Fig. 41.

Bullously enlarged (distended) normal mastoid process.

may occasion flattening or even convex protrusion of the cortical layer into the area of the incisura mastoidea (Figs. 41 and 6). Statistics of examinations of a large number of skulls (Zuckerkandl) have given the following results: pneumatic mastoids were found in

40 per cent., diploic in 20 per cent., and mixed in 40 per cent. of the cases. In the majority of cases, both mastoids of the same individual were of the same internal structure.

V. THE INTERNAL EAR.

The internal ear consists of (1) the membranous labyrinth, with nerve-end organs; (2) the osseous capsule of the labyrinth; and (3) the auditory nerve and its ganglia.

The **membranous labyrinth** develops through ligation of the labyrinthine vesicle from the embryonal epidermis of the parietal region. The embryonal, epithelial, labyrinthine vesicle gradually assumes a more and more complicated shape, and the origin of the completely developed labyrinth from a primary structure, the labyrinthine cyst, can only be understood from the fact that all spaces of the labyrinth are in communication with one another.

Taking the course of development into consideration, we distinguish a pars superior and a pars inferior in the membranous labyrinth (Plate III). The pars superior consists of the utricle and the recess of the utricle (sinus utriculi anterior), superior sinus and inferior, three ampullæ, and three semicircular canals. The ampullæ and the recess of the utricle are invested with nerve epithelium of a characteristic form (Figs. 42 and 43). The nerve-end places of the ampullæ are called cristæ ampullares, and the one situated in the recess of the utricle is called macula utriculi.

The semicircular canals, the ampullæ, and their nerve-end places are named according to their position in the cranium. Thus, there are the frontal (antero-superior), the horizontal (medio-external), and the sagittal (postero-inferior) canals.

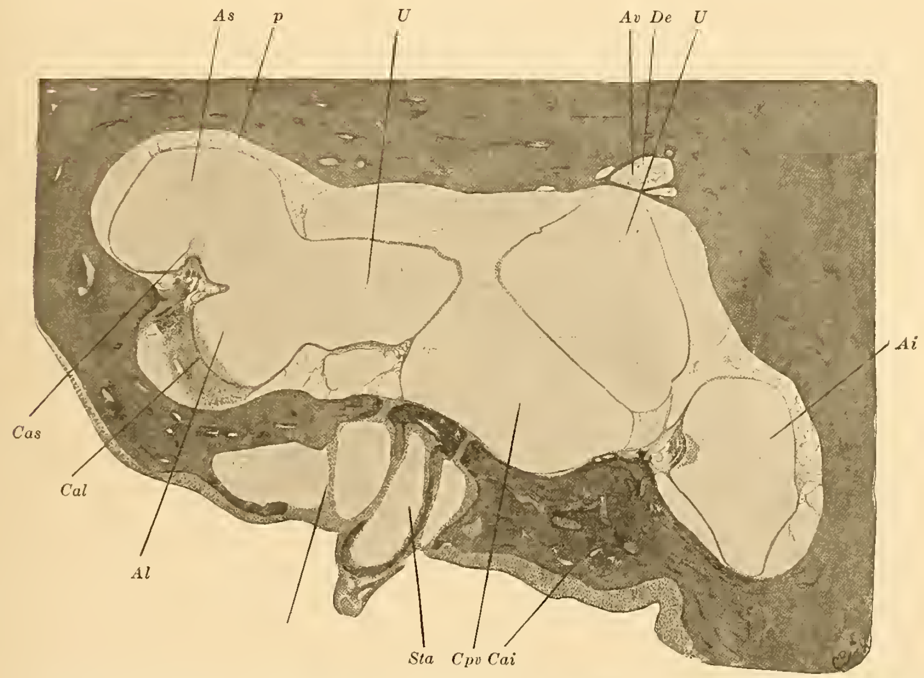
From this the following nomenclature is derived for the ampullæ and the nerve-end places: ampulla superior (frontalis, anterior) and crista ampullaris superior; ampulla lateralis (horizontalis, media) and crista ampullaris lateralis; ampulla inferior (sagittalis, posterior) and crista ampullaris inferior.

The pars inferior comprises the sulcus and the membranous cochlear duct (ductus cochlearis). The latter is funnel-shaped, with its narrow end pointing upward, which communicates through a fine canal (canalis utriculo-saccularis) with the utricle and fuses with the ductus endolymphaticus. This duct proceeds through the vestibular aqueduct to the posterior surface of the petrous bone, where it terminates in an amplified sac (saccus endolymphaticus), enclosed in a kind of pocket formed by the dura. The sac has minute apertures, through which it communicates with the extradural spaces, while in the endodural direction it is completely closed. It connects with the external aperture of

the vestibular aqueduct and often encroaches, downward and outward, for several millimetres upon the medial wall of the sigmoid sinus.

The saccule communicates with the membranous cochlear duct through the ductus reuniens, which inosculates into the cochlear duct, as the small intestine does into the large, forming at the same time a cul-de-sac bordering posteriorly upon the cochlear duct (cæcum vestibulare). The membranous cochlear duct is spirally convoluted (Figs. 43 and 44). Its initial part (vestibular section) is 4 mm. long and is curved on a large radius. The remaining part of the cochlear duct is

Fig 42.



Vertical section through the labyrinth. Topography of the ampullæ. (12 : 1.) *Cas*, crista ampullaris sup.; *As*, ampulla superior; *p*, perilymphatic connective tissue; *U*, utricle; *Av*, vestibular aqueduct; *De*, endolymphatic duct; *Ai*, ampulla inferior; *Cai*, crista ampullaris inferior; *Cpv*, cisterna perilymphatica vestibuli; *Sta*, stapes; *l*, mucous fold of stapes; *Al* ampulla lateralis; *Cal*, crista ampullaris lateralis.

curved on a lesser and gradually diminishing radius and is called the body of the cochlea, while the amplified blind end, which is situated in the apex, is called the cupola (cæcum cupulare). The lumen of the cochlear duct increases from base to apex and has two and three-fourths convolutions in the body of the cochlea. The last quarter of the convolution is formed by the cupola. Stretched out the membranous cochlear duct is about 30 mm. long.

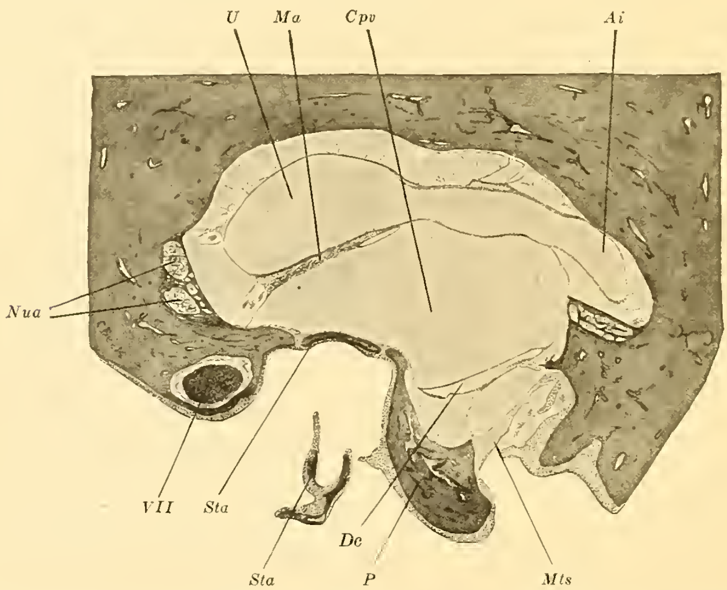
The pars inferior has two nerve terminations: (1) the macula sacculi and (2) the papilla basilaris cochleæ (Corti's organ).

The Nerve Termination of the Labyrinth.—The pars superior of

the labyrinth contains: (1) crista ampullaris superior, (2) crista ampullaris lateralis, (3) crista ampullaris inferior, and (4) macula utriculi. The inferior portion contains: (1) macula sacculi and (2) macula basilaris cochleæ (Corti's organ).

Arranging the above according to the homology of their histological structure, there will be three groups: (1) belonging to the semicircular canal: crista ampullaris superior, lateralis, and inferior; (2) belonging to the vestibule (vestibular sacs): macula utriculi, macula sacculi; and (3) belonging to cochlea: papilla basilaris cochleæ (Corti's organ).

Fig. 43.



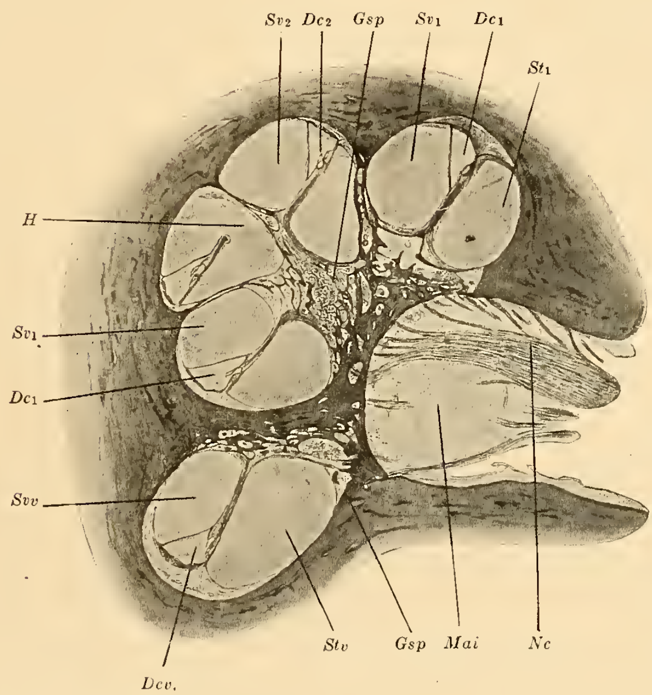
Frontal vertical section through the vestibulum. (10 : 1.) *U*, utricle; *Ma*, macula utriculi; *Cpv*, perilymphatic vestibular cistern; *Ai*, inferior ampulla; *Mts*, secondary membrane of the tympanum; *Dc*, cochlear duct; *Sta*, stapes; *VII*, facial nerve; *Nua*, utriculo-ampullar nerve.

Although there are considerable differences in function and the finer details of structure, there is one uniform ontogenetic basis for these three groups.

At certain places of the embryonal labyrinth, the simple epithelium changes into sensory or nerve epithelium, which comprises two groups of cells: (1) the hair-cells, which are the sensory cells proper; they are barrel-shaped and provided with a number of the finest hair-processes 10–15μ long and 8–12μ wide, arranged like a brush. The protoplasm of each nerve-cell contains the end of a primitive fibre, spirally convoluted or in screw shape, of the nerve bundle to which it belongs (Kolmer). The large cystic nucleus does not communicate with any of the nerve-fibres. The position of the hair-cells in the nerve epithelium is preserved

by specially formed and geometrically arranged prop-cells. There is only one kind of prop-cell in the cristæ and maculæ, while in Corti's organ various prop-cells are differentiated and can be demonstrated in the histological picture. Furthermore, there is one common characteristic in that all the nerve-end places are provided with formed, resistant corpuscles, with which the hair-processes communicate. The hair-processes in the cristæ ampullares and maculæ, and to a certain

Fig. 44.



Axis section through the cochlea of a twelve-year-old child. *Mai*, internal auditory meatus; *Nc*, cochlear nerve; *Gsp*, spiral ganglion; *Dcv*, cochlear duct of the vestibular section; Dc_1, cochlear duct of the basilar convolution; Dc_2, cochlear ductof the medial convolution; *Stv*, scala tympani of the vestibular section; St_1, scala tympani of the basilar convolution; *Svv*, scala vestibuli of the vestibular section; Sv_1, scala vestibuli of the basilar convolution; Sv_2, scala vestibuli of the medial convolution; *H*, helicotrema.

extent also those of Corti's organ, converge toward the median line of the nerve-end places. In the cristæ ampullares they are collected in a cartilaginoid corpuscle (capula terminalis) having the shape of a cap or a saddle and situated at the convex surface of the cristæ. The hair-processes of the macula utriculi and macula sacculi fuse with the otolithic membrane which carries the otoliths.

The cupola as well as the otolithic membrane have indications of a very subtle striation corresponding to the prolongation of the hair-processes. The otoliths consist of calcium carbonate and calcium phosphate; the smallest have a diameter of 2–5μ and are of irregular

or spherical shape; the larger ones have a diameter of 5–25μ and indicate a hexagonal type.

The nerve-end place of the cochlea is covered by the membrana tectoria (cortical membrane), which is homologous to the cupola of the ampullæ and to the otolithic membrane of the vestibular sacs.

The arrangement of the hair-processes and the parts making demand upon the latter indicates that the physiological stimulations of all nerve-end places of the labyrinth are merely dependent upon the causation of movements (pressure changes in the sense of increased or diminished demands, vibrations). These movements are imparted to the hair-processes, and the movements of the latter are the stimulation for the nerve-cells. The stimulation is taken up by the nerve-end fibres inclosed in the nerve-cells, and conducted centrally to the brain.

I. HISTOLOGICAL STRUCTURE OF THE NERVE-END CELLS OF THE LABYRINTH

1. **Histological Structure of the Cristæ Ampullares.**—Each crista ampullaris (Fig. 42) forms a crest bulging into the ampullar lumen vertically to the longitudinal axis. The fissure caused by this position is filled out by the nerve bundles and the connective tissue accompanying the same. The nerve epithelium is bounded against the surrounding cell structure by a line of cylindrical epithelium and a longitudinal sulcus (sulcus cristæ). The vertical section of the cristæ has the shape of a crest or bud, covered by the cupola, which, like the entire crista, protrudes into the free ampullar space.

2. **Maculæ of the Vestibular Sacs.**—Both maculæ are situated at the level of the epithelial wall of the sacs. The macula sacculi lies within the saccule itself, while the macula utriculi lies in a recess of the utricle (recessus utriculi, sinus utricularis anterior). The macula utriculi (Fig. 43) will be seen at the lateral recess wall in the fresh preparation as a white spot in the shape of a heart, and larger than a millet-seed. Exteriorly it is covered by the bundles of the nerve of the utricle. At the endolymphatic surface it carries the otolithic membrane together with the otoliths. The base of the macula utriculi is turned toward the perilymphatic vestibular cistern.

The macula sacculi is smaller than the macula utriculi and forms an oval in the direction of the longitudinal axis of the saccule. It is situated in the middle wall of the saccule, with its base turned toward the osseous vestibular wall.

3. **Papilla Basilaris Cochleæ (Corti's Organ).**—The structure of Corti's organ can only be considered in conjunction with that of the membranous cochlear duct (Fig. 44).

The membranous cochlear duct (Fig. 44, 45) is spread out between

PLATE IV.

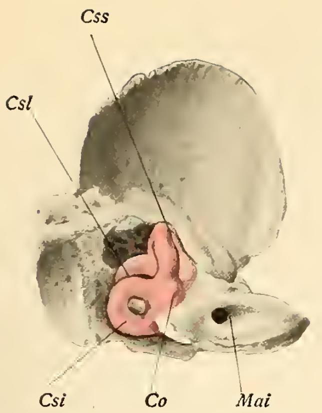


Fig. 1. Osseous labyrinth of a two-months-old child. *Css*, superior semicircular canal; *Csi*, inferior semicircular canal; *Csl*, lateral semicircular canal; *Co*, commissure of the semicircular canal; *Mai*, internal auditory meatus.

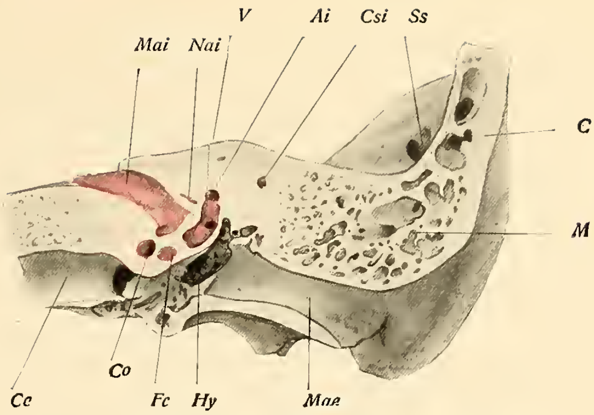


Fig. 2. Horizontal section through the temporal bone of a twelve-year-old child. Inferior half of section. Natural size. *Mai*, internal auditory meatus; *Nai*, canal for nerv. ampullaris inf.; *V*, vestibulum; *Ai*, inferior ampulla; *Csi*, inferior semicircular canal; *Ss*, sigmoid sulcus; *C*, external corticalis; *Mae*, external auditory meatus; *Hy*, hypotympanum; *Fc*, fenestra cochleæ; *Co*, cochleæ; *Cc*, carotid canal.

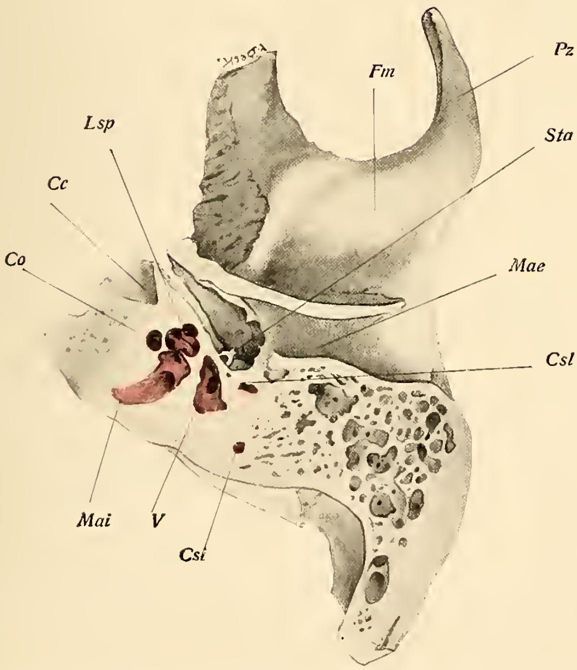


Fig. 3. Horizontal section through temporal bone of a twelve-year-old child. Superior half of section. Natural size. *Cc*, carotid canal; *Lsp*, lamina spiralis; *Fm*, fossa mandibularis; *Pz*, zygomatic process; *Sta*, stapes; *Mae*, external auditory meatus; *Csl*, lateral semicircular canal; *Csi*, inferior semicircular canal; *V*, vestibulum; *Mai*, internal auditory meatus; *Co*, cochlea.

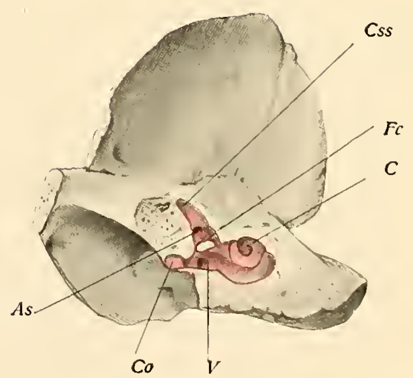


Fig. 4. Lateral wall of labyrinth. Vestibular aspect. One-year-old child. $1\frac{1}{4}$ natural size. *V*, vestibule; *Css*, lateral semicircular canal; *Fv*, fenestra vestibuli; *Co*, commissure of semicircular canals; *As*, superior ampulla.

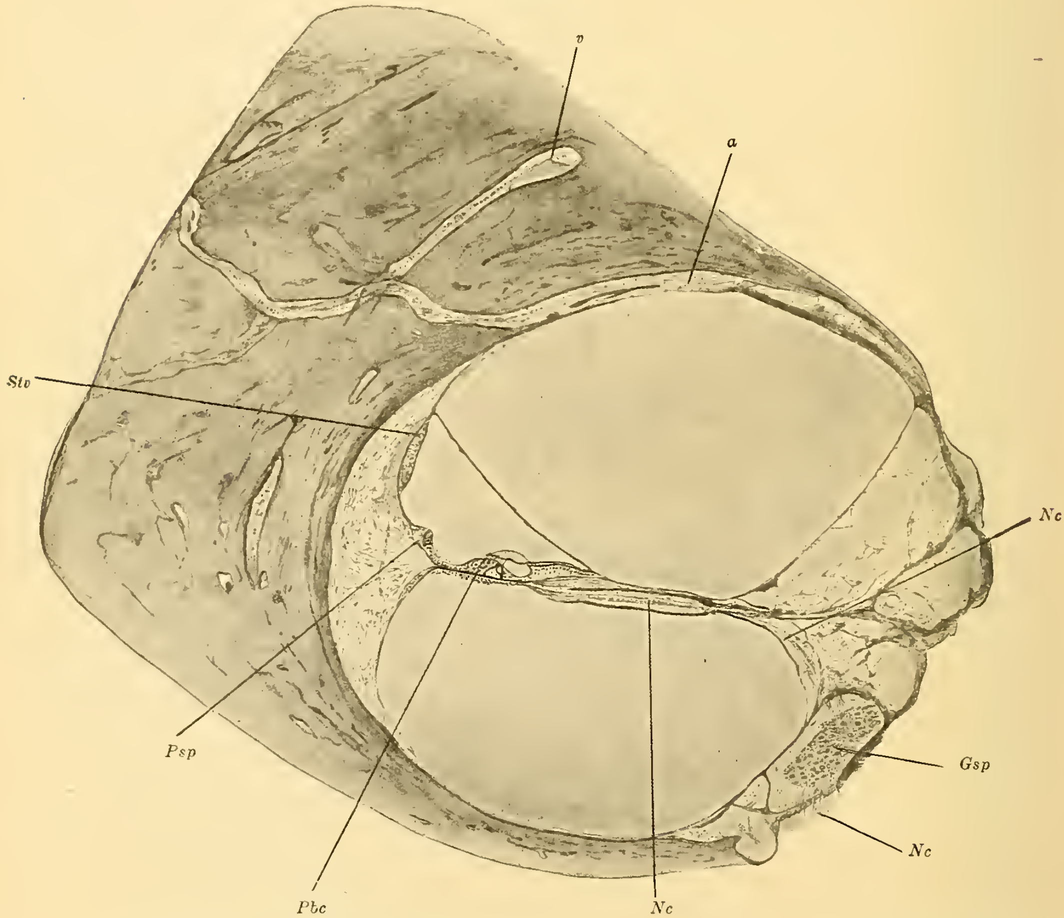
the lamina spiralis ossea and the external osseous cochlear wall. It effects the division of the perilymphatic cochlear space into the scala vestibuli and the scala tympani, being inserted in the endolymphatic canal (which was originally called the scala media). The cochlear duct being axially connected with the osseous cochlear wall more or less in the shape of a ledge, and peripherally in the shape of a plane, it follows that the cross section shows a triangle with an acute axial, an acute superior and an obtuse lateral angle. It is opportune, therefore, in describing the membranous cochlear duct, to distinguish between three walls: (1) the basilar wall (membrana basilaris), (2) the external (peripheral) wall, and (3) the superior wall (membrana vestibularis, Reissner's membrane). The membranous wall is the result of a combination of epithelial (endolymphatic) and mesodermal (perilymphatic) elements of the three distinct walls just described.

4. **Membrana Basilaris.**—The base of the membrana basilaris (Figs. 44 and 45) is formed by most minute, spiral, anuclear connective tissue fibres, which run in an axioperipheral direction (membrana propria). It fuses axially with the crista spiralis, peripherally with the spiral ligament, the ligamentum spirale. The latter is covered toward the scala tympani with a nuclear tympanic membranous layer. Its upper plane, which is turned toward the endolymphatic space, carries the epithelial end-apparatus of the cochlea (Corti's organ) (Fig. 45), which is composed of regularly arranged, polymorphous epithelial cells and consists, like the other labyrinthine end-places, of hair-cells and prop-cells. The radial vertical section of Corti's organ has the shape of a hilly crest. The medial part of Corti's organ is formed by two columnar cells (Corti's columns). Their broad base rests upon the lamina propria, which close against each other at the top in gable-form. At the two sides of the columnar base, which turn toward each other, there are the two basilar cells which morphologically belong to the columnar cells and are supported by Deiters's prop-cells. These are arranged axially from the columns in palisade fashion, with rapidly flattening cylindrical epithelium, which is continued into the cuboidal epithelium of the neighboring sulcus spiralis internus. Peripherally from the columnar cells there are three or four rows of Deiters's prop-cells, and furthermore three kinds of surrounding epithelia: (1) cuboidal epithelium (Böttcher's cells), (2) cylindrical epithelium (cells of Claudius), and (3) high cylindrical epithelium. The latter terminates in convex form and descends from the distal row of Deiters's prop-cells in a semicircle toward the basilar membrane (Hensen's arch). These three epithelial layers are finally followed up by the cuboidal epithelium of the sulcus spiralis externus.

Each row of prop-cells is accompanied by a row of hair-cells, so

that axially from Corti's columns there is one row of hair-cells, while peripherally there are three to four. The hair-processes of the hair-cells are in uninterrupted connection with the membrana tectoria (Corti's membrane) and grow right through the border membrane (membrana reticularis). The primitive fibres, emanating from the external hair-

Fig. 45.



Axis section through the basilar convolution of the cochlea. *Pbc*, papilla basilaris cochleæ; *Psp*, spiral promontory; *Stv*, stria vascularis; *Nc*, nervus cochleæ; *Gsp*, spiral ganglion; *a–v*, anastomosis between the blood-vessels of the petrous bone and the labyrinth (Politzer).

cells, traverse the tunnel space, fuse with the fibres coming from the row of internal hair-cells, and, running through the lamina propria, arrive in the fissure of the lamina spiralis ossea, where they form the cochlear nerve with the aid of the marrow they have acquired.

The crista spiralis is situated on the upper surface of the lamina spiralis ossea in the axial angle of the cochlear duct. It consists of a lower section lined with connective tissue and an epithelial upper section. The

PLATE V.

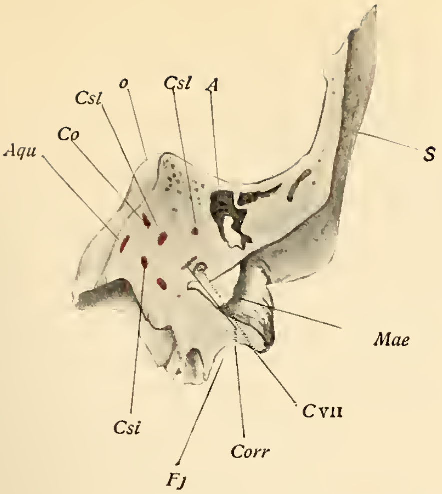


Fig. 1. Frontal section through the nucleus of the labyrinth in the region of the semicircular canals and antrum (*A*). Enlarged 1¼ : 1. *Aqu*, aquæductus vestibuli; *Co*, commissure of the semicircular canals; *Csl*, lateral semicircular canal; *o*, upper edge of petrous bone; *A*, antrum; *S*, squama of temporal bone; *Mae*, external auditory meatus; *C*vii, facial canal; *Fj*, jugular fossa; *Csi*, inferior semicircular canal.

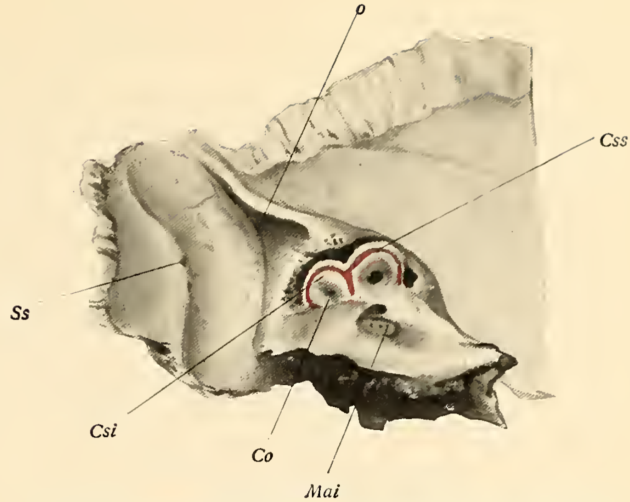


Fig. 2. Topography of the osseous semicircular canals. Left temporal bone of a six-year-old boy. Natural size. *Css*, superior semicircular canal; *Mai*, internal auditory meatus; *Co*, commissure of semicircular canals; *Csi*, inferior semicircular canal; *Ss*, sigmoid sulcus; *o*, upper edge of petrous bone.

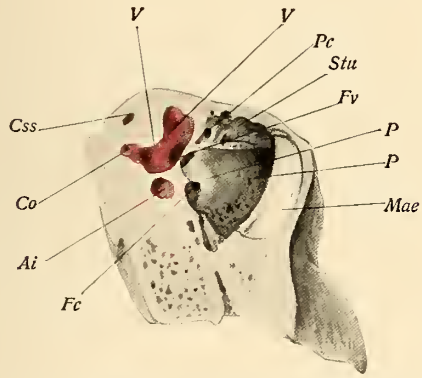


Fig. 3. Window of labyrinth with vestibule. Petrous bone of a six-months-old infant. Frontal section. Enlarged 1.5 : 1. *V*, vestibulum; *Pc*, proc. cochleariformis; *Stu*, semicircularis tubæ; *Fv*, fenestra vestibuli; *P*, promontory; *Mae*, external auditory meatus; *Fc*, fenestra cochleæ; *Ai*, inferior ampulla; *Co*, commissure of semicircular canals; *Css*, superior semicircular canal.

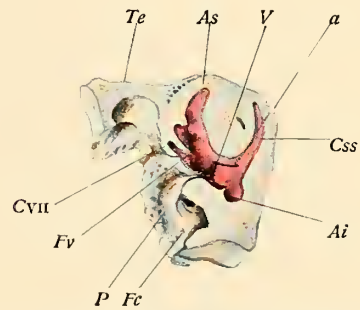


Fig. 4. Medial and posterior walls of vestibule. Vertical section through temporal bone of a six-months-old infant. Enlarged 1¼ : 1. *Te*, tegmen tympani; *As*, superior ampulla; *V*, vestibule; *Css*, superior semicircular canal; *Ai*, inferior ampulla; *Fc*, fenestra cochleæ; *P*, promontory; *Fv*, fenestra vestibuli; *C*vii, facial canal; *a*, sound in the vestibular section of the aquæductus vestibuli.

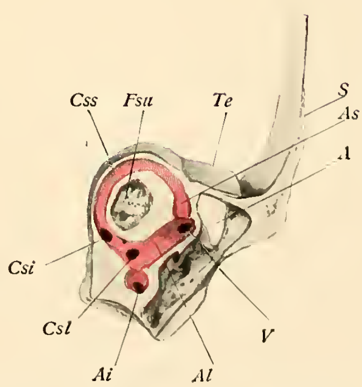


Fig. 5. Posterior wall of the vestibule. Left temporal bone of a six-months-old infant. Vertical section. Enlarged 1¼ : 1. *Fsu*, fossa subarcuata; *Te*, tegmen tympani; *S*, squamous part of temporal bone; *As*, superior ampulla; *A*, antrum; *Al*, lateral ampulla; *V*, vestibule; *Ai*, inferior ampulla; *Csl*, lateral semicircular canal; *Csi*, inferior semicircular canal; *Css*, superior semicircular canal.

latter fuses partly with the epithelium of the sulcus spiralis internus, and partly with the epithelial layer of the membrana vestibularis (Reissner's membrane). Corti's membrane originates at the place where Reissner's membrane separates from the crista spiralis.

5. **The External Wall.**—The base of the external wall (Fig. 45) is formed by the ligamentum spirale. The epithelial wall is independent up to the prominentia spiralis, forming cubicocylindrical epithelium which invests the sulcus spiralis externus. The vascular stria, in which the epithelial and connective-tissue components of the cochlear duct are intimately united, extends upward from the prominentia spiralis to the upper angle of the cochlear duct.

6. **The upper wall of the cochlear duct** (membrana vestibularis, Reissner's membrane) extends between the upper and axial angles (Figs. 44 and 45). It is extremely tender and consists of two layers of flat cells with mostly prominent nuclei. According to embryological development, the inner layer is of epithelial, the external connective-tissue layer of mesodermal origin.

The labyrinth is supplied with blood-vessels by the internal auditory artery. We are indebted to Siebenmann for valuable investigations as to the vascular supply of the labyrinth.

II. THE OSSEOUS LABYRINTH (OSSEOUS CAPSULE) AND THE PERILYMPHATIC TISSUE (PERILYMPHATIC CAPSULE OF THE LABYRINTH)

In describing the osseous capsule of the labyrinth (Plates IV–VII) it was not feasible to adopt the division offered by the membranous labyrinth into superior and inferior sections. The osseous labyrinth should be divided into three sections, which can be done without constraint, as follows: (1) semicircular canals, (2) vestibule, (3) cochlea.

General.

The osseous capsule of the labyrinth develops from a cartilaginous layer which protects the labyrinthine vesicles exteriorly as early as the end of the second month of embryonic life. In the course of development the cartilaginous layer grows over the labyrinth with an increasing curvature, enveloping it toward the auditory nerve, and, while thus advancing toward the future internal auditory meatus, forms the cartilaginous capsule of the labyrinth. The osseous capsule of the labyrinth is the result of ossification of the cartilaginous embryonal capsule. In the new-born it is still to a certain extent independent of the other parts of the petrous bone and isolated through surrounding cartilaginous remnants (islands) and large marrow spaces. The latter sometimes persist until the second year, and the cartilaginous islands are under

certain circumstances still present until the thirtieth year; nevertheless, intimate connections of the osseous compacta of the capsule with that of the petrous bone always occur at an early period, so that in the majority of cases there can only be a question of cavities of the osseous labyrinth, but not of a disconnected osseous labyrinth from the second year onward. It is only exceptionally that the petrous bone is pervaded for a greater length of time by large cavities (marrow spaces or pneumatic cells), thereby more or less isolating the osseous capsule.

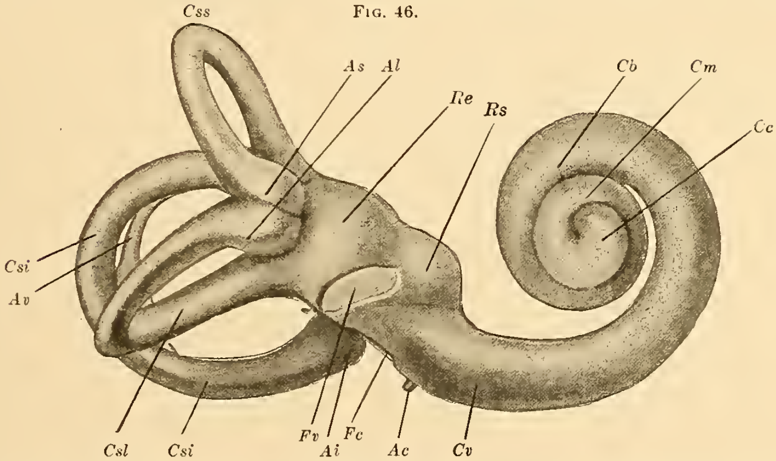
The Vestibule.—The vestibule is a roomy, empty space of irregular formation (Plates IV–VII). For purposes of description, it is convenient to assume a cuboidal form with six walls. The lateral wall contains the vestibular window, which is occluded by the plate of the stapes and leads into the middle ear (Plate IV, Fig. 4). Closely above the vestibular window and next to the lateral pole there is the hook-shaped end of the crista vestibuli, which runs over the upper and anterior walls toward the inferior one, where it gradually diffuses. The crista vestibuli is divided by the vestibulum into a large, superior (recessus ellipticus) and a small, inferior part (recessus sphæricus). The upper wall (Plate IV, Fig. 3) and the inner wall (Plate V, Fig. 4) of the vestibulum have no apertures whatever, while the anterior wall (Plate V, Fig. 3) contains the area cribrosa sacculi. It is situated in the centre of the recessus sphæricus, which is destined to receive the saccule. Medially, the slit-like terminal aperture of the vestibular aqueduct adjoins the recessus sphæricus (Plate V, Fig. 4). The aqueduct terminates in the vestibulum with a tangent aperture, in about the same way as the ureter communicates with the bladder. The posterior wall of the vestibulum (Plate V, Fig. 5) contains the terminal aperture of the lateral ampulla and the lateral semicircular canal. At the juncture of the posterior and superior walls, the terminal ostia of the upper ampulla and the commissure of the semicircular canal (the common crus of the superior and posterior semicircular canals) are situated.

The terminal aperture of the ampulla of the posterior sagittal semicircular canal is situated at the lower plane of the vestibulum. The crista vestibuli diffuses at the lateral margin of the ampullar ostium, while in the external part of the lower plane the vestibular base unites with the primary and secondary spiral osseous layers of the cochlea. The vestibular cul-de-sac of the membranous cochlear duct is situated in a fossula of the vestibulum. Right in front of this place the fissure commences which, bounded by both laminæ spirales osseæ, effects the communication of the scala tympani with the scala vestibuli, as can be seen in the macerated preparation. This fissure is bridged over and closed by the membrana basilaris of the membranous cochlear duct. The area cribrosa superior is visible at the upper end of the crista ves-

tibuli. The latter forms the passage-way for the fibres of the utriculo-ampullar nerve to leave the bone, and, after these fibres have traversed the vestibulum itself for a short distance, they arrive at their nerve-end places.

The Osseous Semicircular Canals.—The position of the osseous semicircular canals (Plates IV–VII) coincides with that of the membranous canals which terminate in them. The diamater of the osseous canals is about eight times greater than that of the membranous ones, while the circumference of the osseous ampulla exceeds but slightly that of the membranous ampulla. The consequence is that the osseous canals are less sharply marked against the osseous ampullæ than the membra-

FIG. 46.



Cavity of the bony labyrinth. *Css*, canalis semicircular superior; *Csl*, canalis semicircular lateralis; *Csi*, canalis semicircular inferior; *As*, ampulla superior; *Al*, ampulla lateralis; *Ai* ampulla inferior; *Av*, aquæductus vestibuli; *Re*, recessus ellipticus; *Rs*, recessus sphæricus; *Fv*, fenestra vestibuli; *Fc*, fenestra cochleæ; *Ac*, aquæductus cochleæ; *Cv*, cochlea—vestibule; *Cb*, cochlea—basal turn; *Cm*, cochlea—second turn; *Cc*, cochlea—gyrus semitertius.

nous canals are against the membranous ampullæ. The average figures in the new-born are the following: diameter of the osseous canals 1.25 mm., length of frontal canal 12 mm., length of horizontal canal 13 mm., length of sagittal canal 14 mm.

The Osseous Cochlea.—The osseous cochlea (Plates IV, VI, VII) consists of the following parts: vestibular section (6 mm. long), body of cochlea, apex of cochlea. The vestibular part is the connecting tube between vestibulum and body of cochlea.

The body of the cochlea has 2½ convolutions and terminates in a flat cupola. The radius of curvature diminishes from the cochlear base to the apex. The convolutions are differentiated as basal, medial, and apical, and are separated from one another by osseous walls. The nerve-fibres coming from Corti's organ diffuse in the axial part of the cochlea (modiolus) and reach the internal auditory canal through the tractus

spiralis foraminosus (Fig. 45). There is a double lamella (lamina spiralis ossea primaria), originating from the modiolus at the median height of each convolution, which receives the nerve-fibres coming from Corti's organ. The lamina spiralis divides every cochlear convolution into an upper (scala vestibuli) and a lower compartment (scala tympani). The interposition of the membranous cochlear duct completes the separation. The lamina spiralis ossea primaria is confronted with the secondary osseous spiral lamella (lamina spiralis ossea secundaria) in the vestibular section and in the lower half of the basal convolution, both lamellæ uniting as they pass into the lower wall of the vestibulum. At the upper end of the medial convolution, the lamina spiralis ossea loses its line of insertion at the modiolus, and protrudes in the shape of a spirally curved, pointed hook (hamulus) into the apical convolution (Plate VI, Fig. 5; Fig. 45). The aperture hereby occasioned between the cochlear axis and the hamulus (helicotrema) allows the two compartments to communicate axially from the membranous cochlear duct. Resection of the peripheral cochlear wall from the modiolus exposes the wall at the end of the cochlear cupola as a lamellar terminal surface of the modiolus, and this part of the external wall of the cupola is called the lamina modioli. The cochlear aqueduct, which opens into the lower surface of the petrous bone, originates in the scala tympani near the cochlear window. It forms a connecting tube of the perilymphatic spaces with the intradural space, into which it opens.

The nerve-fibres in the lamina spiralis ossea are spiral, and arranged in a backward direction. The ganglionic duct (Rosenthal's duct) branches off in the modiolus, following the spiral course of the lamina spiralis ossea.

III. TOPOGRAPHY OF THE NERVE-END PLACE OF THE LABYRINTH
(Plates III, VI, VII)

The surfaces of the cristæ ampullares form, similarly to the semicircular canals and ampullæ, a right angle against each other. Corresponding to the wall of the sulcus, the macula utriculi and the macula sacculi are slightly convex. Constructing a medial plane for both will show the following result: with erect position of the head, the macula sacculi appears approximately frontal and vertical, the macula utriculi runs vertically against the macula sacculi, slightly descending from outward and upward to inward and downward.

IV. TOPOGRAPHY OF THE INTERNAL EAR

The membranous labyrinth is parietally fixed in the spaces of the osseous labyrinth (Figs. 42–45). This fixation is accomplished by the perilymphatic connective tissue which invests the osseous surface with an endosteal layer and the membranous labyrinth with a subepithelial

layer, the perilymphatic ligaments extending between both layers. The spaces of the osseous labyrinth are not completely filled at any place by the membranous labyrinth. While at some places there are only cleft-like perilymphatic interspaces (at the ampullæ, the upper wall of the utricle, etc.), there are at other places large perilymphatic spaces which in extent far exceed the regional endolymphatic spaces. Such spaces are the following:

(1) Along the semicircular canals: perilymphatic canals.

(2) In the vestibule in the projection of the lateral vestibular wall and stapes: the vestibular cistern (cisterna perilymphatica vestibuli).

(3) In the cochlea in the shape of cochlear canals.

The perilymphatic spaces of the semicircular canals are isolated from the vestibular cistern by connective-tissue layers. The scala vestibuli is distended at the vestibular end of the cochlear duct, reaching into the vestibular cistern and communicating at the apex of the cochlea with the scala tympani through the helicotrema. The lower cul-de-sac of the scala tympani is situated at the membrana tympani secundaria. The facial nerve (Plate VII, Fig. 1) terminates superficially in the internal auditory meatus in its S-shaped course. The utriculo-ampullar nerve lies below and travels outward and backward. The sacculo-ampullar nerve sends an isolated branchlet into the canaliculus ampullæ inferior. The cochlear nerve occupies the lowest position and runs forward and downward.

The endolymphatic spaces communicate with each other, also with the lymph fissures of the dura through the vestibular aqueduct, and indirectly with the extradural spaces of the posterior cranial fossa. The perilymphatic spaces are in direct communication with the intradural spaces through the cochlear aqueduct and through the cleft-like lymph spaces along the nerve branches of the internal auditory canal.

In infants and children up to the end of the second year many parts of the osseous labyrinth extend to the surface of the petrous bone, as, for instance:

1. The external crus of the lateral semicircular canal protrudes at the middle of the antrum wall;

2. The vestibular section of the cochlear duct is situated below the promontory.

3. The eminentia arcuata corresponds to the vertex of the superior semicircular canal, the fossa subarcuata to the hollow space bounded by the same canal (Plate V, Fig. 5).

4. The commissure of the semicircular canals is distinctly recognizable at the posterior surface of the petrous bone.

5. The upper surface of the sagittal semicircular canals protrudes crest-like at the posterior surface of the petrous bone.

6. The upper part of the basal cochlear convolution causes a flat-convex eminence at the superior petrous surface.

7. The external apertures of the two aqueducts and of the hiatus spurius of the facial canal lie at the superior petrous surface.

The osseous layer covering the osseous labyrinth increases in density in proportion to the increase in size of the petrous bone, as development proceeds. The development from the time of puberty onward proceeds as follows:

Topographically important places which remain unchanged are the prominence of the lateral semicircular canal and the promontory. The vertex of the frontal semicircular canal, which in the new-born is often covered with an osseous lamella so thin and transparent that the membranous canal may often be seen through it in the fresh preparation, is far away from the eminentia arcuata in the adult, sometimes as far as several millimetres.

Commissure, sagittal canal, and superior crural pole are far removed from the upper surface of the petrous bone and covered with a thick compact bone. The ostia of the hiatus spurius and vestibular aqueduct are grown over by osseous lamellæ which impart a cleft-like form to the canal-shaped ostia. The cochlear aqueduct is considerably increased in length and has formed a gaping triangular ostium. The internal auditory canal is considerably elongated in the course of growth. The fundus of the auditory canal either remains unchanged or becomes amplified, while the external aperture of the internal auditory canal (porus acusticus internus) is usually narrower than in the new-born. The total post-embryonal increase in size of the labyrinth is about 18 per cent.

V. EIGHTH NERVE

The fibres of the eighth nerve (Plates III and VII) terminate in the hair-cell protoplasm of the labyrinthine nerve-end places. The peripheral branches of the auditory nerve are interrupted by a ganglionic mass composed of bipolar cells. This mass forms three vestibular ganglia,—superior, inferior, and spiral.

The utriculo-ampullar nerve terminates in the upper vestibular ganglion, the sacculo-ampullar nerve in the lower vestibular ganglion, and the cochlear nerve in the spiral ganglion. The latter is situated in the canalis ganglionaris of the cochlea; the two vestibular ganglia, which are centrally connected with each other, are situated deep in the internal auditory canal. The union of the fibres emanating from the vestibular ganglia separates the eighth nerve centrally from the ganglia into two branches (roots), the vestibular and the cochlear. The nervus intermedius, which is situated in the internal auditory meatus over the vestibular nerve, does not communicate with the eighth and terminates

PLATE VI.

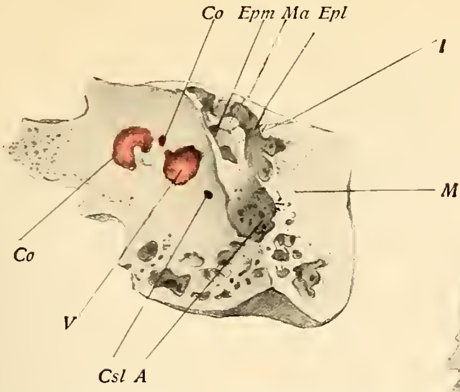


FIG. 1. Attic (*Ep*), vestibule (*V*), antrum (*A*), and cochlea (*Co*). Right temporal bone of a fourteen-year-old child. *Co*, cochlea; *Epm*, medial attic; *Epl*, lateral attic; *I*, incus; *Ma*, malleus; *M*, mastoid process; *A*, antrum; *Csl*, lateral semicircular canal; *V*, vestibule.

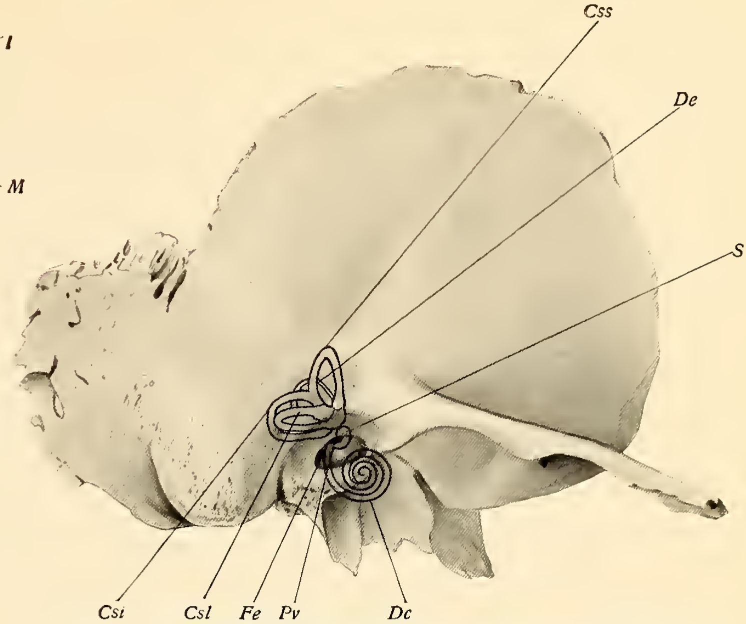


FIG. 4. Topography of labyrinth (external aspect). Enlarged 1¼ : 1. *Fc*, cochlear window; *Pv*, vestibular section of cochlear duct. For further abbreviations see Fig. 2.

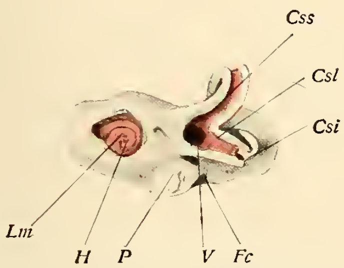


FIG. 5. Labyrinthine nucleus of petrous bone of a three-year-old child. Spaces of labyrinth opened from without. Enlarged 1¼ : 1. *Css*, superior semicircular canal; *Fc*, fenestra cochleæ; *Csl*, lateral semicircular canal; *V*, vestibule; *Csi*, inferior semicircular canal; *P*, promontory; *H*, helicotrema; *Lm*, lamina modioli.

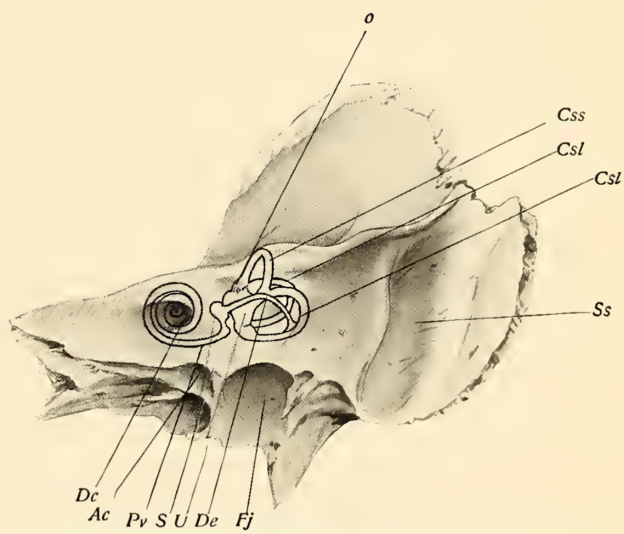


FIG. 3. Topography of labyrinth (posterior aspect). Enlarged 1¼ : 1. *Ac*, cochlear aqueduct; *o*, superior edge of petrous bone; *Fj*, jugular fossa. For further abbreviations see Fig. 2.

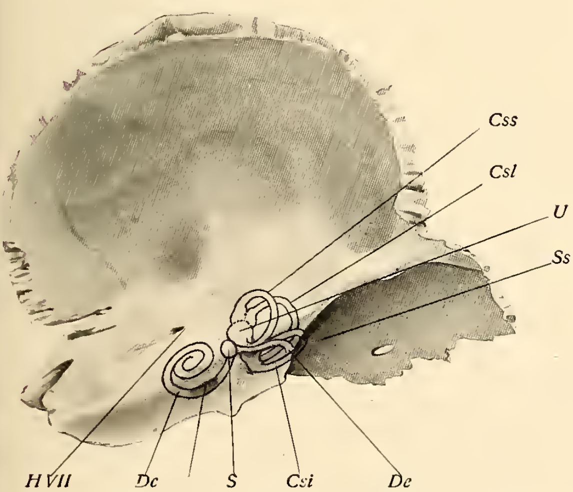


FIG. 2. Temporal bone of a two-year-old child. Projection of the membranous labyrinth (in superior aspect). Enlarged 1¼ : 1. *HVII*, Hiatus spurius of the facial canal; *Css*, superior semicircular canal; *Csl*, lateral semicircular canal; *U*, utricle; *Ss*, sigmoid sulcus; *De*, ductus endolymphaticus; *Csi*, inferior semicircular canal; *S*, saccule; *Dc*, cochlear duct.

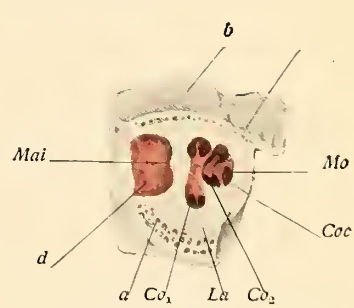


FIG. 6. Modiolus (*Mo*). Enlarged 1¼ : 1. *b*, cerebral surface of petrous bone; Co_1, basal convolution; Co_2, medial convolution; *Coc*, apical convolution; *La*, labyrinthine nucleus (compact bone); *a*, spongy paralabyrinthine zone; *d*, canaliculus ampullæ inferioris; *Mai*, internal auditory meatus.

in the geniculate ganglion. Joined with the facial, the acoustic arrives at the crus cerebri at the lateral margin of the pons. The vestibular nerve runs medially from the restiform body to the medulla oblongata. Its descending part (radix of the spinal acusticus) extends down to the region of the hypoglossal nucleus and terminates in the small-celled vestibular nucleus (nucleus parvicellularis vestibularis). Deiters's nucleus, which is also called the nucleus of coördination, is besides intimately related to the cerebellum, the nerve-nuclei of the ocular muscles, and the motor nerves of the nuchal musculature.

The vestibular, centrally from its nucleus, passes through the substantia reticularis into the optic thalamus. The further course of the vestibular nerve is not definitely known.

A direct communication of the fibres of the vestibular nerve with the cerebral cortex has not been demonstrated, but it is assumed that its vestibular course, after one or two interruptions, terminates in the posterior portion of the cortex of the parietal lobe (Munk's sensory sphere). It is also assumed that this cortical region plays a part in space orientation.

The cochlear nerve arrives laterally from the restiform body in the crus cerebri and the ventral and dorsal cochlear nuclei (nucleus accessorius and tuberculum acusticum). The cochlear fibres traverse centrally the fourth ventricle and, after crossing the median line, play the principal part in the formation of the lateral loop. Part of the fibres terminate in the superior olivary body. The centripetal course of the cochlear nerve is again interrupted in the looped nucleus, in the posterior corpus quadrigeminum and the geniculate ganglion. The central end runs across the posterior part of the inner capsule in the cortex of the temporal lobe (probably the first temporal convolution, gyrus transversus). The striæ acusticæ arise from the tuberculum acusticum and take partly a homolateral, partly a central course, crossing each other.

Each temporal lobe is supplied from both auditory nerves. In central affections of the cochlearis, therefore, there can never be a question of unilateral deafness, as the affection, whether partial or total, must necessarily be bilateral.

II. PHYSIOLOGY OF THE EAR

Two sensory apparatuses are combined in the function of the anatomical entity of the ear,—the auditory organ in a physiological sense to perceive the sound, and a sensory organ for orientation in space, perception of position, of accelerations, and indirectly for the maintenance of the body equilibrium. This sensory organ is called the static organ (Breuer) or static labyrinth.

The sound-perceiving organ is the papilla basilaris cochleæ (Corti's organ), which is situated in the cochlea. It enables a person of normal hearing to perceive a great variety of sounds within the scale of high and low limitation, noises, and mixtures of sounds and noises. The lower acoustic limit of the tuning-fork is 12–16 (double) vibrations, the upper one 35,000 to 40,000. The sound vibrations are carried by the air from the sound source into the ear.

Sound is conducted to the cochlea in three ways:

(1) *Air conduction*, in which we distinguish meato-tympanic and pharyngo-tympanic conduction. The meato-tympanic route appeals most to the auditory sense. The sound waves pass through the external auditory meatus into the tympanic cavity by means of the tympani membrane and the chain of auricular ossicles, and thence into the cochlea. The efficacy of the pharyngo-tympanic route has been demonstrated by Politzer's experiments showing that the sound waves pass through the Eustachian tube into the tympanic cavity and thence into the cochlea as in the meato-tympanic conduction.

(2) *Bone Conduction.*—The sound waves originating in the air and those produced by directly applied vibrating bodies impart the vibrations to the cranial bones, whence they are transmitted to the cochlea. There are a cranio-tympanic and a cranio-labyrinthine conduction, according to Bing. In the former the waves imparted to the bone are retransformed into air waves in the tympanic cavity, whence they reach Corti's organ in the same way as in air conduction. In the cranio-labyrinthine conduction the waves imparted to the cranial bones exert a direct effect upon the cochlea without being transformed in the tympanic cavity. In bone conduction sound is normally perceived only through the cranial bones, although it has been observed that young individuals may sometimes perceive sounds from bones further distant (scapula, radius, etc.).

(3) *Cartilage conduction*, which represents a certain form of bone conduction.

PLATE VII.

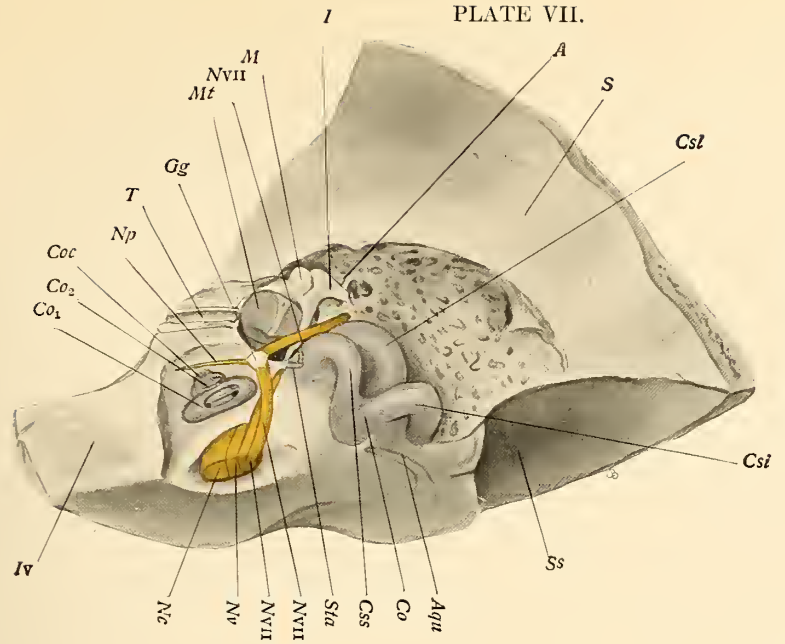


FIG. 1. Topography of the ear. *Mt*, tympanic membrane; *N*VII, facial nerve; *M*, malleus; *I*, incus; *A*, antrum; *S*, squamous part of temporal bone; *Csl*, lateral semicircular canal; *Csi*, inferior semicircular canal; *Css*, superior semicircular canal; *Co*, commissure of semicircular canal; *Aqu*, aquæductus vestibuli; *Ss*, sigmoid fossa; *Sta*, stapes; *Nv*, vestibular nerve; *Nc*, cochlear nerve; *Iv*, impressio trigemini; Co_1, basal convolution; Co_2, medial convolution; *Coc*, apical convolution; *Np*, large superficial petrosal nerve; *T*, tuba auditiva; *Gg*, geniculate ganglion.

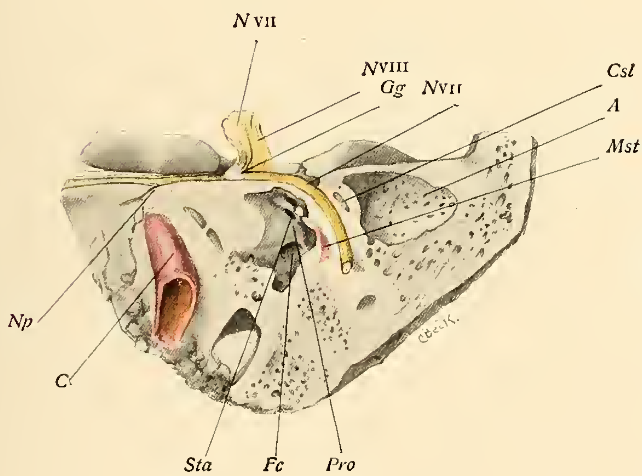


FIG. 2. Topography of the facial nerve. *N*VII, facial nerve; *N*VIII, auditory nerve; *Gg*, geniculate ganglion; *Csl*, lateral semicircular canal; *A*, antrum; *Mst*, stapedius muscle; *Pro*, promontory; *Fe*, fenestra cochleæ; *Sta*, stapes; *C*, carotid; *Np*, large superficial petrosal nerve.

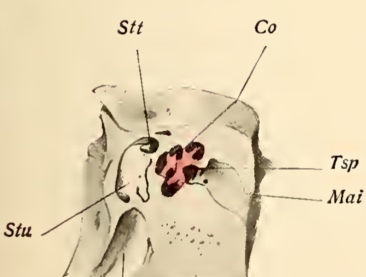


FIG. 3. Topography of the cochlea (*Co*). *Tsp*, tractus spiralis foraminosus; *Mai*, internal auditory canal; *CC*, carotid canal; *Stu*, semicanalis tubæ; *Stt*, semicanalis tensoris tympani.

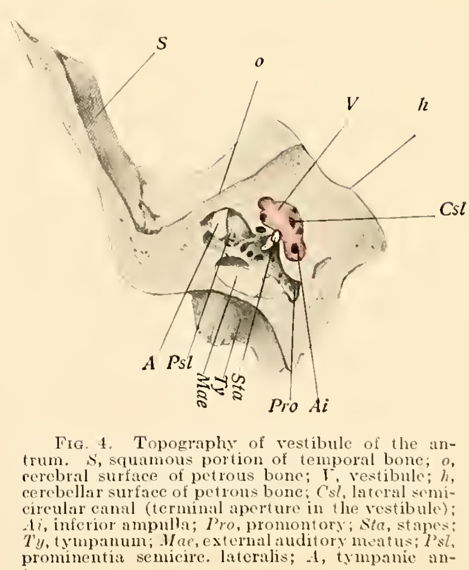


FIG. 4. Topography of vestibule of the antrum. *S*, squamous portion of temporal bone; *o*, cerebral surface of petrous bone; *V*, vestibule; *h*, cerebellar surface of petrous bone; *Csl*, lateral semicircular canal (terminal aperture in the vestibule); *Ai*, inferior ampulla; *Pro*, promontory; *Sta*, stapes; *Ty*, tympanum; *Mae*, external auditory meatus; *Psl*, prominentia semicirc. lateralis; *A*, tympanic antrum.

THE THEORY OF HEARING. THE PHYSIOLOGY OF THE SOUND-CONDUCTING APPARATUS

According to Helmholtz's theory, or hypothesis, of resonance, the end-organ within the cochlea is capable of analyzing a sound impression into its component parts. Acoustico-analytical properties of this kind are also possessed by string instruments, and especially by the piano. We may confidently assume that the cochlea as well as the piano has a number of strings of varying size which are attuned to certain sounds. The assumption that the fibres of the basilar membrane are indeed attuned to certain sounds is confirmed by the fact that they are of different lengths in different parts of the cochlea. The basilar fibres of the cochlea, 15,000 to 20,000 in number according to Hasse-Retzius, increase in length from base to apex.

The crucial point in Helmholtz's theory is the supposition that a certain sound causes only certain strings to vibrate, or a certain group of sounds a certain group of strings. These strings are always those which, according to their length, are attuned to the sounds conducted to the ear. The string fibres of the cochlea being enclosed in the basilar membrane, there can be no question of vibration of single fibres, but only of certain radial portions of that membrane. Helmholtz's theory easily and completely explains the phenomenon of floating sounds, secondary sound manifestations, the sound gaps and sound islands observed in many cases of oral affections and deaf-mutism. It pays due regard to intermediate sounds, combination, differential, summation, variation and intermittent sounds.

Wundt has expressed the opinion that each sound reaches the central organ in two ways: (1) through the cochlea, for which he adopts Helmholtz's theory, and (2) through a direct incitement of the fibres of the auditory nerve by vibrations imparted to the cranial bones, circumventing the cochlea, which means by way of skull conduction. Wundt's opinion is based upon Ewald's communication on the hearing capacity of animals without labyrinths (pigeons) and on a number of observations made on patients with unilateral destruction of the labyrinth. These observations, however, have not withstood criticism, and to-day it is considered an established fact that a sound-impression can only lead to physiological excitation of the auditory nerve, and thereby conscious perception, by way of the cochlea and Corti's organ.

Hermann attributed to the ear the ability of perceiving each period as sound. Later he adopted Helmholtz's theory, adding, in order to explain intermittent sounds, that each string fibre and each definite section of Corti's organ was only in connection with a definite nerve-cell (Zahlzelle). But since it was demonstrated that the intermittent

sounds are either of physical origin or else differential sounds, his cell theory has become superfluous (Schäfer).

An independent theory of hearing has been proposed by Max Meyer. The vibrations imparted to the cochlea by the middle ear are transmitted to a shorter or longer piece of the basilar membrane according to the objective intensity of the sound, which would be subdivided into a number of sections vibrating more or less rapidly. Each definite section corresponds to a definite sound, the length of the section determining the subjective intensity of the sound, and the frequency of vibrations its position in the scale. A similar idea of static waves in the basilar membrane is contained in the theory of ter Kuile. Finally, Ewald thinks that each sound imparted to the ear causes the basilar membrane to vibrate along its entire length, subdividing the same into a number of static waves. The perception of sound is effected by the totality of these static waves,—Ewald's sound picture. Each sound has a characteristic sound picture. Ewald has based his views upon a large number of excellent and exact observations on vibrating rubber membranes. In order to do the greatest possible justice to the anatomical conditions, he constructed rubber membranes the size of the cochlear basilar membrane and graphically reproduced his sound pictures.

THE SENSE OF HEARING IN THE NEW-BORN

The ability of hearing in the new-born is very deficient. The external auditory meatus is not permeable, and the spaces of the middle ear are nearly filled with gelatinous tissue. The surface of the tympanic membrane is uneven, and the membrane itself is thick.

Sound conduction is, therefore, considerably interfered with both in the external and middle ear. Such an impediment occurring in the adult from diseased conditions would considerably diminish the auditory acuity, reducing it to perhaps one or two metres in ordinary conversation, and this distance will probably represent the auditory acuity of the new-born.

Motor reaction upon sound-perceptions in the new-born has been demonstrated during the first two weeks, the earliest time recorded being the fourth day (Preyer).

Normal auditory acuity develops in the course of the first few weeks with the gradual resorption of the connective-tissue deposits in the middle ear and the progressive development of the membranous auditory meatus into a freely permeable, air-filled passage.

It is an important requirement for the normal function of the ear that all the spaces of the middle ear be full of air and that the pressure in these spaces entirely coincides with the pressure from without. The normal mucous membrane of the tympanic cavity absorbs small quan-

tities of oxygen and it is necessary, therefore, from time to time to renew the air present in the middle-ear spaces, which means ventilation. This ventilation is effected by the reflex or intentional act of deglutition, which opens the Eustachian tube and allows air to penetrate through it into these spaces. In the absence of reflex deglutition or in pathological changes of the tube which prevent its opening in deglutition, there will be considerable changes in the tympanic membrane and interference with hearing owing to partial or complete resorption of the air in the middle-ear spaces.

THE PHYSIOLOGY OF THE STATIC LABYRINTH (APPARATUS OF THE SEMICIRCULAR CANALS AND VESTIBULE)

Taking into consideration the cellular structure of the nerve-end places of the labyrinth, the method of their arrangement, and the fact that they are provided with active factors (cupola, otolithic membrane, Corti's membrane), it will be understood that the physiological stimulation for all the nerve terminations must necessarily consist in motion (molecular motion, waves, vibration, oscillation, percussion). Corti's organ is only responsive to acoustic waves and, accordingly, admits of vibratory motion in its position in the cochlea. Any such contingency is out of the question for the vestibular nerve terminations (macula utriculi, macula sacculi) as well as for the ampullar ones (cristæ ampullares).

Corti's organ serves the perception of sound, and the mere consideration of the anatomical structure of the cristæ and maculæ forces us to the conclusion that the vestibular and ampullar nerve terminations are not expected to transmit the perception of sound.

Flourens's discovery, in 1828, that experimental injury of the semicircular canals in pigeons was followed by vertigo and disturbances of equilibrium, was of fundamental importance for a knowledge of the function of the vestibular apparatus. Ménière, in 1861, found apoplectic vertigo and equilibrial disturbance to have been caused by a hemorrhage of the labyrinth. Kreidl, in 1887, experimentally demonstrated the influence of gravitation upon the otolithic apparatus of crabs. Verworm, in 1891, published his important investigations on equilibrium and the otolithic organ of the ktenophoræ. Karl L. Schaefer demonstrated, in 1894, that invertebrate animals (without semicircular canals) do not suffer from rotatory vertigo, and that the latter does not occur, in frog larvæ for instance, until the semicircular canals have completely developed. Alexander and Kreidl, in 1901, found that congenital arrest of equilibrium in the dancing mouse was caused by anomalies of development in the vestibular sacs and their nerves.

Theories on the Functions of the Labyrinth

The first to elaborate a useful theory, on the ground of anatomical and experimental facts, was Goltz. According to his theory, the nerve terminal organs of the semicircular canals are organs of equilibrium, although it does not entirely exclude the participation of the labyrinthine nerve-end places in the function of hearing. The introduction of the conception "organ of equilibrium" is due to Goltz's theory.

According to Goltz, there is an independent ciliating motion of the hair-processes which entertain endolymphatic waves.

Ewald, in his interpretation of the function of the semicircular canals, agrees with Goltz and speaks of "Goltz's sensory organ." Besides, however, he assumes the existence of a second otolithic (statolithic) apparatus. According to Ewald, they both serve in the perception of movements of the head and body, and indirectly the maintenance of equilibrium. The most important part of Ewald's theory, however, is the establishment of the conception of the "tonus labyrinth." The normal labyrinth, according to this theory, has an influence upon the tonus of the entire voluntary musculature, including the stabile equilibrium of the entire body, and the conception of the tonus labyrinth leads without restraint to an understanding of the muscle reflexes due to the labyrinth.

Ewald's theory of looking upon the labyrinth as an organ for maintaining or regulating the tonus is undisputed, and the idea of the labyrinth playing a part in maintaining or controlling the equilibrium can be simply and clearly deduced from its tonus-regulating effect.

Delage looks upon the labyrinth as an organ for the perception of position and movements of the head, a theory which branches over to that of Mach-Breuer. The latter has been carefully built up on the basis of anatomical and experimental facts. Accordingly, the semicircular canals are organs for the perception of positive and negative angle accelerations, while the otolithic apparatus (vestibular apparatus in the narrower sense) is intended for the perception of positive and negative straight-lined accelerations. The organism is, therefore, tri-axially provided for through the three semicircular canals in regard to angular acceleration, while straight-lined accelerations are only bi-axially cared for by the two maculæ of the vestibular sacs; and this refers not only to man, but to all terrestrial mammals. Swimming and flying animals possess a third nerve terminal with otoliths, which is situated at the peripheral end of the cochlear duct in a section homologous to the vestibular sacs (lagæna and macula lagænæ).

It was Breuer who proposed the now generally accepted term of "static labyrinth" to designate both the semicircular canals and the vestibular sacs.

Before entering upon the question of physiological function of the normal human labyrinth in ordinary life, it is necessary to explain why it is difficult to understand this sensory apparatus and to demonstrate its function.

There are three causes:

(1) The higher organs of special sense are distinguished by easily recognizable functions which are strictly differentiated. So far as the lower organs of special sense are concerned, there are transition stages. Thus, under given circumstances, it may be difficult to decide whether a certain perception is due more to the organ of taste or of smell. On the other hand, the perceptions emanating from the static labyrinth, whatever theory may be adopted, possess no characteristic points which could not, at least partially, be transmitted in a similar form by other organs of special sense, especially the eye, and also by artificial or deep sensations (skin, muscle, articulation). We are, therefore, compelled to remove the labyrinthine kernel from the entity of perceptions by special methods of observation or special arrangements of examination. We have to recognize and exclude all perceptions transmitted by the eye as well as superficial and deep sensations.

(2) The second cause for the difficulty in demonstrating the functions of the labyrinth lies in the fact that the vestibular nerve has no direct communication with the cerebral cortex. The perceptions conducted centrally through the vestibular nerve become our conscious property mostly or exclusively by way of reflexes and their psychophysiological sequelæ.

(3) Finally, the following point presents itself for consideration: The various pathological conditions of the static labyrinth must be deduced from the various forms of the reflex excitability of the labyrinth, and we have consequently to distinguish between the normal, the pathologically exaggerated, the pathologically diminished, and arrested (negative) excitability of the static labyrinth.

The Functions of the Normal Apparatus of the Semicircular Canals and Vestibule at Physical Rest and in Motion

Delage's fundamental experiments have shown that the perception of the body position or orientation on the perpendicular is not materially changed if all skin and articular sensations are excluded so far as may be possible (firmly tying down in oblique position). In diving, the upward force of the water counteracts the gravity of the body. Nevertheless, we retain a distinct and correct perception of up and down under water with closed eyes and without tactile orientation. Both observations lead to the assumption of the presence of a special sensory organ regulating

the equilibrium. Further experiments compel us to look for this organ in the head and finally lead to the recognition of its identity with the static labyrinth. In proof of this assumption, James's experiment has been frequently cited, in which disorientation was observed in deaf-mutes under water. It would be very necessary, however, to subject these observations to further tests.

At the instigation of M. Sachs, R. Bárány and myself examined the psycho-physiological significance of the statolithic apparatus for normal individuals and deaf-mutes as to orientation in space, and found that there is no difference between these two classes as to the recognition of the perpendicular. It may be assumed that the stimulations of the statolithic apparatus are only of slight importance, if any, for forming a conception of the perpendicular in normal individuals.

It is chiefly through the communication of the vestibularis with the cerebellum that the static labyrinth is enabled to act as an organ for maintaining and controlling equilibrium. The preservation of the normal muscular tonus is equivalent with normal stability of the human body. The centre of perception of motion and position, however, is not situated in the cerebellum, but in the cerebrum—in the foci of the central convolutions (Munk's sensory sphere). With these, however, the vestibularis is only in indirect communication.

Generally speaking, affections of the semicircular canals cause such manifestations as are otherwise elicited by experimental stimulation, such as nystagmus, vertigo, and disturbances of equilibrium. Our present knowledge of the symptomatology of the affections and destruction of the semicircular canals is already considerable, but we know much less about the symptoms due to isolated affection of the vestibular apparatus. These consist of equilibrial disturbances without vertigo, impairing the stability of the body. The destruction of the entire labyrinth, perhaps even the destruction of the vestibular sacs, leads to changes in the tonus of the body musculature. In cases of this kind there are hallucinations of labyrinthine character. Thus, we are justified in speaking of ocular disturbances contingent upon the function of the labyrinth, and the disturbances of equilibrium following upon optic and motor phenomena are referable to a reaction upon the labyrinth. Finally, there are disturbances of the sense attitude to be considered, such as feeling larger or smaller, heavier or lighter.

Coördination and subordination are matters whose regulation presupposes activity on the part of the vestibular apparatus.

III. EXAMINATION OF THE EXTERNAL AUDITORY CANAL AND MIDDLE EAR

1. OTOSCOPIC EXAMINATION

If the conditions for observation are favorable and the auditory canal is of ample width, it may be possible to survey the entire length of the latter and the tympanic membrane without any further preparation or instruments. To institute a methodical examination of the external and internal ear, however, a reflector and ear-speculum are necessary. The reflector consists of a concave mirror, perforated in the centre, of 7.5 cm. diameter and 16 cm. focal distance. An oval aperture for inspection is preferable to a round one. The reflector is fastened with a band or an elastic metal rim over the operator's forehead, but may also be attached to a frame or support which is held with the teeth. The ear-specula are made in various shapes and sizes, either from hard rubber (Politzer) or metal. Gomperz has introduced a very short speculum for otoscopic examination of infants during the first weeks of life, which does excellent service. White cloud-light is sufficient for purposes of examination. Among the artificial light sources, the uncovered Auer lamp is still the simplest and best. The lamp is placed behind the patient slightly above the head, and the ear to be examined is turned away from the light.

The first step is to illuminate the entire aural region by means of the reflector. If there are no changes at the auditory meatus, the concha is gently drawn backward and upward, and the speculum inserted with a slight rotatory movement until it reaches the border line between the cartilaginous and osseous canals, or in the new-born until the upper margin of the tympanic membrane becomes visible. While the speculum is held in this position with the left hand, it is arranged with the right so that all or some parts of the tympanic membrane may be inspected. The instruments necessary for examination are also handled by the right hand.

2. METHODS OF CLEANSING THE EXTERNAL CANAL AND THE TYMPANIC CAVITY

In order to obtain a sufficiently clear otoscopic picture, it is necessary to remove the normal or pathological contents of the external auditory canal and tympanic cavity (cerumen, epidermal crusts, pus, blood, etc.), as otherwise free inspection of the tympanic membrane or the parts behind it, if perforated, is interfered with. An angularly flexed button-probe serves for examining these masses, which can be

removed by small forceps. It is advisable to have two sizes of ear-pincettes in readiness which are carefully grooved at the ends (Politzer's ear-pincettes). The pus is removed with cotton tips, which have been sterilized in sterilizers or by singeing over a flame. At the first examination, irrigation with a syringe is preferable to the removal of pus with a cotton tip. We are using a sterilizable syringe which can be taken apart, holding from 150–200 c.c. It is fitted with a number of cannulæ of different sizes. In syringing the ear, the external meatus is drawn up and back, as is done in the otoscopic examination, and the cannula is gradually inserted from behind and upward for about 5 mm. In order to widen the auditory duct, the mouth should be held wide open. Weak antiseptic solutions (2 per cent. boric or salicylic acid, or sterilized water) at a temperature of 100°–104° F. are used for irrigation. Sublimate solutions are not permissible in children.

To cleanse the middle-ear spaces, we are using Politzer's glass cannulæ. These are provided with a rubber tube and bulb, and by aspiration or compression a liquid instilled into the auditory duct and tympanic cavity (5 per cent. peroxide, alcohol, etc.) can be moved to and fro. The cleansing process should be commenced with aspiration, introducing the cannula with bulb compressed. The irrigation fluid generally used is peroxide 5.0, aq. dest. 30.0.

The cleansing process may be extended further by the use of Hartmann's attic cannula. Generally speaking, I prefer metal cannulæ which can be sterilized by boiling, but in the case of infants and children it may sometimes be advantageous to use hard rubber or flexible attic cannulæ, made of soft red rubber or silk rubber. The latter are gradually inserted under the guidance of the speculum down to the place it is intended to irrigate, and it is advisable to leave the manipulation of the bulb, containing the irrigation fluid, to an assistant. Syringing or irrigating the ear for diagnostic purposes may be combined with testing the labyrinth for caloric excitability.

3. NORMAL OTOSCOPIC PICTURE OF THE EXTERNAL AUDITORY CANAL AND TYMPANIC MEMBRANE. OTOSCOPY OF THE TYMPANIC CAVITY

The posterior end of the exterior auditory canal is sharply demarcated in the otoscopic picture against the tympanic membrane by an angle and by the color, the canal having the normal color of the skin, while the tympanic membrane is tinged gray or grayish-blue (pearl-gray).

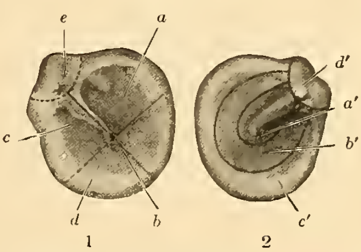
In the normal otoscopic picture (Figs. 24, 47) the entire tympanic cavity can be surveyed. Anteriorly from above toward the centre of the membrane is the manubrium; the posterior fold of the tympanic membrane extends from the short process toward the posterior margin

of the membrane. In normal conditions of the middle-ear spaces it is hardly visible in infants. The anterior and posterior folds are usually entirely absent. The radial fibres of the tympanic membrane, accumulated at the umbo, sometimes form the pes anserinus.

The circular fibrous bundles stretch along the tympanic margin of insertion and are usually not visible in the otoscopic examination of infants. The light-reflex (light-cone, light-sector, light-spot) extends from the middle of the tympanic membrane anteriorly and inferiorly, and is most intense at its central point.

The division of the tympanic membrane into quadrants and circular sections (Fig. 47) will facilitate the description of the pathological findings. Dividing the membrane into quadrants, the pars tensa comprises the anterior, superior and posterosuperior, anterosuperior, and postero-inferior quadrants. The pars flaccida (Shrapnell's membrane) forms a special, fifth section.

Fig. 47.



Topographical division of the tympanic membrane. 1. Division into quadrants: *a*, posterosuperior quadrant; *b*, postero-inferior quadrant; *c*, anterosuperior quadrant; *d*, antero-inferior quadrant; *e*, membrana flaccida. 2. Circular division: *a'*, central part of the tympanic membrane; *b'*, medial part of the tympanic membrane; *c'*, peripheral part of the tympanic membrane; *d'*, membrana flaccida of the tympanic membrane.

The circular sections divide the membrana tensa into a central, medial, and peripheral (external) part of the tympanic membrane. A fourth part is supplied by the membrana flaccida.

According to the position of the tympanic membrane in the skull, the posterosuperior quadrant is nearest the eye of the examiner, and this part is also normally larger than the others.

The gray or grayish-blue tint of the tympanic membrane is much less due to the color of the membrane itself than to the law of turbid media seen through the air enclosed in the tympanic cavity behind the membrane.

The parts of the tympanic cavity or its contents which are closest to the membrane are, in many cases, visible through the membrane like shadows. These parts are (*a*) the long crus of the incus, (*b*) the promontory, (*c*) the recess of the cochlear window.

If other parts of the tympanic contents become transparent, such as the stapes, chorda tympani, folds of the malleus, etc., it is a sign of pathologically increased translucency of the tympanic membrane due to atrophy.

If the auditory canal markedly kinks downward, it may be impossible with the ordinary speculum to survey the entire extent of the antero-inferior quadrant. This would necessitate the application of a specially thin speculum which would freely expose the inferior quadrant.

Otherwise the width of the speculum should be closely adapted to the dimensions of the external auditory canal. If the speculum is too narrow, it will not be in contact with the walls of the canal, and an inexperienced operator may find it difficult, if not impossible, to localize the tympanic membrane. If it is too large, the movable integument of the membranous auditory canal will be pushed forward in folds, which would considerably interfere with the distinctness of the picture.

If the tympanic membrane is completely absent, it is possible to survey the greater part of the internal wall of the cavity, the hypotympanum, the mesotympanum, a considerable part of the contents of the cavity, and part of the walls of the attic and antrum. In this way it may be possible to investigate otoscopically the entire middle-ear spaces down to the tympanic orifice of the tube. In small defects of the tympanic membrane, examination of the perforated fundus with the probe and irrigation of the middle ear may directly or indirectly supply information on the condition of the middle-ear spaces. Excellent accessory instruments are the attic speculum (Urbantschitsch) and specula with a magnifying glass. Hegener has devised an instrument with which it is possible to examine the tympanic membrane with the binocular stereoscope.

The electric otoscope, now on the market, offers numerous advantages, including ease of manipulation, uniform illumination and, by means of the rotating lens, considerable magnification of the field.

The best of these instruments has a bulb attached so that the tympanic membrane may be moved, or, if the membrane be perforated, secretion may be aspirated through the opening.

IV. METHODS OF EXAMINING THE MIDDLE EAR

1. EXAMINATION OF MOBILITY OF THE TYMPANIC MEMBRANE AND OF THE CONTENTS OF TYMPANIC CAVITY

The mobility of the tympanic membrane and the chain of auricular ossicles is tested by means of Siegle's otoscope. A tube, about 3 cm. long and 1.5–2 cm. in diameter, is attached to an ear-speculum and closed at the other end by a glass plate at an inclination of 45°; laterally an air-tight connection is made with a tube carrying a bulb. Inspection of the tympanic membrane, while the bulb is being operated, renders it possible to decide whether the tympanic membrane has normal, exaggerated, or diminished mobility. Atrophied places bulge out upon aspiration, while adherent parts remain immovable and thickened parts show slight mobility. If the membrane is perforated, the fluid contents of the cavity (serum, pus) may be aspirated through Siegle's instrument into the auditory canal and evacuated. Before inserting the tube, it is advisable to heat the glass plate slightly over a non-smoking flame so as to ensure its continuous clearness.

2. THE VALSALVA TEST.

If the nares are compressed, with mouth closed, a jerky expiratory effort will condense the air in the nasopharyngeal space, and, as the condensed air penetrates into the middle ear through the passively opened Eustachian tube, the tympanic membrane will outwardly protrude to its maximal extent. In the otoscopic picture the manubrium is seen vertically downward and the posterosuperior quadrant appears still larger. The outward displacement of the tympanic membrane is felt by the patient as a jerk in the aural region or as a subjective noise (crackling). The auscultation tube (otoscope) will serve to establish this noise objectively.

If the Eustachian tube is occluded from acute or chronic swelling of the mucous membrane, spasms or tubal musculature, or strictures, or if the walls of the tube are interagglutinated by mucous exudates, the Valsalva test will be negative, which means that the expiratory pressure is not sufficient to open the tube. In that case it will be necessary to establish the kind and degree of tubal affection by other physical methods.

The Valsalva test is very important for the demonstration of perforations of the tympanic membrane, especially in cases of traumatic rupture, the air escaping outwardly through the perforation after passing the tube and tympanic cavity. The noise is established by auscultation.

The tone level of the noise depends upon the size of the rupture. In large gaps there is a deep, bellowing noise, and the smaller the rupture and the more it inclines to the shape of a fissure, the more will the sound approach to a high whistling noise. Crepitant râles point to accumulation of secretion in the middle-ear space. According to Kugel's suggestion, the air escaping through the rupture in the Valsalva test may be conducted through water and demonstrated by the ascending air bubbles.

In perforation of the tympanic membrane, the Valsalva test will be positive only if the tube is quite permeable.

Patients suffering from catarrh of the middle ear usually come to recognize the Valsalva test by accident when blowing the nose. At first they feel considerable improvement, which will lead them to repeat the process frequently. As a rule, they soon acquire considerable aptitude in carrying out the experiment, and are able to exercise the expiratory pressure necessary for the opening of the tube in a manner almost imperceptible to others. The unavoidable consequence of unlimited repetition of this procedure invariably leads to atrophy of the tympanic membrane (Politzer).

3. POLITZER'S METHOD (AIR DOUCHE, INSUFFLATION AFTER POLITZER)

The patient holds a swallow of water in his mouth. A small rubber tube, attached to a bulb, is inserted into the proximal nostril. The right hand holds the bulb, the left compresses the nostrils tightly over the tube. While the patient performs the act of deglutition, and at the moment the larynx ascends and the velum palati closes the œsophagus downward, the air is expressed into the nasopharyngeal space. The overpressure hereby produced in the latter space causes the Eustachian tube to open and allows the air to flood the middle-ear spaces.

It is usually impossible to make young children hold the water in their mouths or to swallow instantly on command. As a substitute they may be ordered to pronounce a word ending with a guttural (such as chuck, clock). In other cases, a sipping inspiration may bring about a successful result. In infants it is best to wait for the commencing expiration in crying, during which it is possible to carry out successfully the insufflation.

If it is intended to give a full air douche, the bulb is compressed in powerful jerks with the entire hand; a less powerful effect is obtained by compressing the bulb with the flat fingers or a few fingers only. It is advisable to have children blow their noses before commencing proceedings. If the permeability of both tubes is normal, the air douche will affect both ears simultaneously. If there is considerable resistance from occlusion, Politzer's method may be unsuccessful, so that the air condensation obtainable in the nasopharyngeal space is not sufficient to

overcome the impediment. If the permeability of only one tube is affected, the air may escape through the healthy tube, and in order to prevent exaggerated bulging out of the tympanic membrane on that side, the auditory meatus is firmly closed with a moistened finger.

The air douche is carried out three or four times at one sitting through the right and left nostrils alternately, so as not to injure the sensitive integument of the nares and the mucous membrane of the nasal septum. Should there be any fissures at the nares, the rubber tube should be greased with borovaseline before insertion.

Politzer's method of insufflation is exceedingly valuable for both diagnostic and therapeutic purposes. Of diagnostic importance are the improvement in auditory acuity and the changes occurring in the otoscopic picture of the tympanic membrane. The penetration of the air into the tympanic cavity is established by auscultation, the operator connecting his ear with that of the patient by the otoscope. The success of an insufflation is also indicated by murmurs, caused by the vibrations of the velum palati. Sometimes it is impossible to avoid some of the injected air passing into the stomach through the œsophagus when the occlusion is removed, but this will cause no inconvenience and the air will escape again in a few minutes. Atrophied portions of the tympanic membrane or cicatrices may be torn if the air is injected with undue force. If the tympanic membrane is perforated, there will be a characteristic noise which is audible in Politzer's method by auscultation. If the Eustachian tube is of large diameter and the defect of the tympanic membrane considerable, the escape of air is sometimes directly felt by the examiner through the otoscope. Up to the time of puberty, Politzer's method represents the only reliable examination of the middle ear and is, besides, of considerable therapeutic value.

4. CATHETERIZATION

The catheter cannot be used in children until they are from 10 to 14 years old. It is especially indicated in chronic middle-ear affections in which air douches are attended with negative results owing to pathological obstacles in the tube. The narrowness of the lower nasal entrance at an earlier age and the slight extension of the nasopharyngeal space are unfavorable to the introduction of a catheter. Its use in older children may become necessary in defects of the hard palate or in paralysis of the velum palati.

The ear-catheter is introduced into the tubal aperture through the lower nasal canal. An elastic, well-polished, smooth hard rubber catheter (Politzer) with an oval aperture is preferable to one made of German silver. Of course, the latter can be sterilized by boiling, but the hard rubber catheters can also be reliably sterilized in a sublimate

solution and be kept thoroughly clean by rinsing with sterilized water. They are preserved dry.

The use of the catheter should be preceded by a rhinoscopic examination. Should the latter reveal any constricting changes in the nose, the catheter is introduced into the nostril under the guidance of the reflector and the nasal speculum. The operator connects his ear with that of the patient through the otoscope.

As soon as the front end of the catheter has passed the posterior border of the hard palate, the problem is to direct the downward aperture of the catheter into the opening of the pharyngeal tube. The most convenient way to do so is to describe a quarter screw-like turn, backward, outward, and upward. Other topographical methods to facilitate the search for the tubal opening or ridge are the following:

(*a*) The catheter is advanced to the posterior wall and introduced into Rosenmüller's fossa by turning the beak outward by 90°; withdrawing the catheter makes it jump over the tubal ridge into the opening. Both operator and patient distinctly feel the catheter as it elastically glides over the tubal ridge.

(*b*) The catheter is inserted through the lower nasal canal in the usual way and advanced to the posterior faucial wall. The beak is then turned toward the opposite side until it is in a horizontal position, and withdrawn to the posterior margin of the nasal septum. From this position the beak is turned by 180° downward, where it will find the tubal ostium.

Both methods are unpleasant for the patient; contact with the sensitive faucial mucous membrane, the velum palati, and the posterior margin of the nasal septum may cause nausea, retching, and even positive vomiting.

If the nasopharyngeal mucosa is inflamed and swollen, the catheter, even if ever so carefully handled, may cause injury to the mucous membrane with subsequent hemorrhage. This is always a very unpleasant incident, for, although the hemorrhage is not severe, it may last for hours. Disagreeable and long-lasting hemorrhages may also occur in the lower nasal canal from injuries to the small vessels of the septum by careless or rough insertion of the catheter.

When the catheter has reached the tubal ostium, the beak is turned upward, so that its curvature may assume the direction of the tubal axis. This is accomplished by turning the ring of the catheter ("fly") upward toward the lateral angle of the canthus. The catheter is now firmly held at the nostril by the first and second fingers, the little finger finding support against the forehead of the patient. The operator next connects bulb and catheter with his free hand, gently and carefully compressing the bulb. Powerful insufflation may be made only after

the characteristic auscultatory noise has convinced the operator that the beak is correctly situated in the tubal orifice.

If the catheter is in a wrong position or if there is an injury to the mucous membrane, incautious insufflation may lead to acute emphysema of the nasopharyngeal mucosa or of the entire facial epidermis. Such an emphysema, however, does not signify any danger, and will usually disappear after a few hours or at the most in a few days, when the air has been resorbed. Still, traumatic emphysema is less a sign of inexperience than of careless and even rough handling of the catheter.

Catheterizing is done with the patient in a sitting posture. If the operator prefers the erect position, he should carefully avoid bending his body forward or exercising any unnecessary pressure on the instrument, as it would give the patient unpleasant and painful sensations at the tubal orifice in the fauces. The catheter should glide along the base of the nose during insertion. If the nasal half pertaining to the ear to be examined should not be permeable, catheterization may be done through the other half or through the mouth with a catheter of greater curvature. It would be far preferable, however, to desist from the process altogether for the time being, and remove the nasal obstruction by operation, especially as this is usually the cause of the aural affection.

Insufflation may be carried out with a simple rubber bulb, a duplex bulb, a compression pump, or bellows.

In marked stricture of the Eustachian tube, bougies of whalebone, celluloid, gold or silver may be used. A special Eustachian catheter is introduced in the usual way, inflation attempted, then the bougie, which must be passed with considerable care, otherwise the mucous membrane may be lacerated. Recently electric bougies have been used. Obviously, this procedure demands knowledge and skill.

V. THE FUNCTIONAL HEARING TEST

The function of the auditory organ is to perceive both sounds and noises. Formerly sounds and noises were considered separate perceptions, but stroboscopic examination has demonstrated the fact that all kinds of acoustic productions belong together. According to former notions, the cochlea could only perceive sounds, while the rest of the labyrinth (semicircular canals and vestibule) perceived noises; but the fact is now established that the last-named parts of the aural apparatus have nothing to do with the act of hearing, and that acoustic manifestations of whatever nature are perceived by the cochlea alone. The stimulation excited in the cochlea is transmitted to the cerebral cortex by the auditory nerves and their central tracts.

To make an exact hearing test, it is absolutely necessary for the patient to be perfectly alert and to exert a marked degree of concentration and attention. It is not sufficient for the patient to react more or less automatically to sound stimulation; he is expected to make definite statements, as for instance where he has heard a certain sound, or to indicate by signs at what moment certain sounds of the tuning-fork cease to be audible; he has to differentiate sounds and to state his perception as to their quality and timbre.

In the test for bone conduction, patients should be requested to separate psychically the sensation of vibrations of the tuning-fork from the perception of the sound and to pay exclusive attention to the latter.

I. DETERMINATION OF AUDITORY ACUITY IN RELATION TO SPEECH; THE ACOUMETER

If the auditory acuity is to be tested by having certain sounds repeated, it is essential that the patient should not merely hear the words, but understand them and be intelligent enough to repeat them.

It is unavoidable, therefore, that hearing tests embrace to a large extent intellectual tests, and for this reason exact and comprehensive hearing tests in infancy are surrounded with difficulties. The age limit coincides about with the school age, but prolonged tests should not be made on children under 10 years of age, since their power of concentration is too rapidly exhausted and reliable statements of perceptions may not be expected. Even in adults, especially in nervous persons, a few minutes' interval must frequently be allowed in order to exclude the possibility of inaccurate statements due to fatigue.

In pathological examinations, the functional hearing test follows after the otoscopic examination. The external auditory canal should

be cleansed and any accidental obstacles to sound conduction, such as cerumen, epidermal parts, scales, pus, etc., removed, so that the functional test may give a clear picture of the capacity of the organ.

The functional test is best divided into one for auditory acuity (distance) for spoken sounds, and one for noises.

The production of spoken sounds is dependent upon: (1) articulation (function of the vocal muscles), (2) condition of the vocal cords, (3) expiratory pressure under which the air escapes from the lungs while speaking.

Articulation is the same in all kinds of speech. The ordinary tone of conversation is obtained by articulation with tension of the vocal cords in ordinary expiration. Very loud conversation requires forced expiration. Articulation with relaxed vocal cords produces whispering. If this relaxation is associated with the maximal pressure of expiration, the result will be accentuated whispering. Typical whispering is produced with the aid of residual air which is simply allowed to pass out from the lungs. Residual air is that quantity of air which remains behind in the lungs after ordinary expiration.

The normal hearing distance for exaggerated conversational sounds in the open is about 60 yards; for ordinary conversational language and under average conditions of conduction in closed rooms, 40–50 yards; for accentuated whispering, 25–30 yards; for typical whispering, 20–25 yards. These figures refer to absolutely normal organs, and will undergo a considerable reduction when the organs are even slightly pathologic.

Importance of the Hearing Test by Speech.—The importance of the hearing test by speech, especially by conversation, requires no demonstration. If a person complains of defective hearing, he means in the majority of cases that he cannot catch the spoken sound, and it is clear therefore that, aside from other tests, the auditory acuity for speech should be especially determined. Besides, whatever improvement in the auditory faculty is achieved by treatment must be tested by the perception of the spoken sound.

The exaggerated conversational tone is only used where the aural affection has so far advanced as to render the perception of ordinary conversation impossible. It serves to find out whether patients can still understand words or only perceive sounds. The hearing distance for exaggerated conversation need not be ascertained, since overloud conversation at a distance of one-half yard only occurs in cases where ordinary conversation is not understood.

It is the latter which is of fundamental importance for the hearing test. It represents that kind of speech which is almost exclusively practised in ordinary life, and should always be taken into consideration whenever the hearing capacity of a patient in his intercourse with others

is to be established. The hearing distance for ordinary conversation, however, is so great that in a clinical room of average size the limitation of slightly reduced capacity cannot be tested. In these cases typical whispering is resorted to, and from the results conclusions are drawn as to the patient's ability to understand ordinary conversation. Accentuated whispering is of less importance in clinical examinations, since it may be heard at a comparatively great distance and is unsuited to determine the limitation of hearing in slight affections even in comparatively large clinical rooms (5 × 6 yards). If, however, the hearing distance is to be established for definite performances, such as are prescribed by law, for instance, accentuated whispering is the only test to be applied, because here individual differences of speech on the part of various examiners as to distinctness and quality of sound are least pronounced.

TEST ARRANGEMENT FOR SPEECH

Binaural Test.—The patient is placed opposite the physician, with his eyes cast down. Correct answers to simple questions, adapted to his range of understanding, will establish the hearing distance. This test yields only an approximate, but very characteristic, picture of the hearing ability of the patient, and it is advisable always to use the binaural test at the first examination as well as at the close of the treatment.

As a matter of course, certain precautions should be observed, to which further reference will be made in the description of the monaural test.

Monaural Test.—In the monaural test the hearing capacity of the right and the left ear is established separately by excluding the function of one ear so far as possible. But it may at once be stated that a complete functional exclusion of one ear is impossible, thus rendering an ideal monaural test illusory. It is impossible to prevent a certain quantity of sound waves reaching the other ear by conduction of the cranial bones, a point which will be further discussed when treating of unilateral deafness. The patient closes the meatus of the ear not to be examined by inserting a moistened finger tip into the canal, care being taken not to press the tragus against the orifice of the canal. In examination of young children this should be attended to by a third person. The other hand is held before the patient's eyes so that he may not be able to read off or guess the words from the examiner's lips.

Method.—The patient turns the ear to be examined toward the physician. This is the "direct method," and the established distance is called the auditory distance by direct method. When the closed ear is turned toward the physician, we speak of the "half-direct method," and when the physician stands with his back toward the closed ear, it constitutes the "indirect method." Other expressions are direct, half-direct, and indirect conversation.

Computation.—In order to find the distance for direct conversation, the figure obtained for half-direct conversation is increased by one-third, that obtained by the indirect method by two-thirds. For instance, a hearing distance of 6 yards in half-direct conversation is equal to 8 yards of direct conversation, or 6 yards of indirect is equal to 10 yards of direct conversation. In this way it is possible to find out the hearing acuity of a person up to 10 yards in a room only 6 yards long, or up to 15 yards in a room 9 yards long. If the patient is able to repeat correctly and promptly the required words at the greatest distance attainable in a given room, the findings are marked by +. Thus C+10 would mean that the patient has correctly and promptly repeated ordinary "Conversation" at a distance of 10 yards.

Field of Combination. Medial Hearing Distance.—The hearing distance for conversational language can only be ascertained as "medial distance." A patient may faultlessly and promptly repeat all test words at a distance of 6 yards, while at a distance of 7½–8 yards he may not be able to repeat a single word. On the other hand, at a distance of 6–7½ yards it is found that he can still correctly repeat easy, polysyllabic words, words containing high vowels and the consonants "r" and "s" and adapted to his range of intellect and thought. His hearing at this distance is indistinct; he takes in only the "word-skeleton" and combines with surprising rapidity some word from the sounds he has caught. Sometimes this is the correct word, but the evidence of its being the result of combination is furnished by the fact that he does not repeat the word instantly, but only after a few seconds' meditation. In most cases, however, the resultant word, though similar in sound, is totally different in meaning, as, for instance, "paper" for "vapor," "window" for "widow," etc.

Correct combination is more than usually easy in the repetition of numerals, and it is therefore inadvisable to use numerals exclusively for test purposes, as is recommended by some authors.

The distance at which a patient hears only the word-skeleton and constructs the supposedly correct word by combination is called the combination distance or field. Its extent chiefly depends upon the patient's age and temperament. Children under 10 years of age and phlegmatic or torpid individuals but rarely exhibit any combination field. In these cases the positive hearing distance immediately borders upon the negative field, in which not a single word can be repeated. Active individuals combine to a much greater extent than the phlegmatic; furthermore, patients with a chronic aural affection resort to combination more frequently than those suffering from an acute affection, for the simple reason that the former are constantly compelled in

ordinary intercourse to resort to it and have thus acquired greater practice. In this connection it is a surprising fact that the gift of combination is entirely independent of the degree of intelligence, quite uneducated individuals sometimes furnishing a very interesting and varied field of combination.

How to Test by Speech.—In establishing the medial hearing distance, care should be taken to exclude all sources of error in order to expedite the test. The words should be so spoken that the patient may distinctly hear and understand them. Wolf, of Frankfort, was the first to divide the German language into word groups of varying audibility. Words of a high degree of audibility are those containing several syllables and high vowels; those of medial audibility are polysyllables with high and dull vowels; and to this group belong the numerals. Words of bad audibility are those of one or two syllables containing dull vowels and labial sounds.

The audibility of a word is increased by pronouncing it slowly and distinctly; this is facilitated by preceding each substantive by the article and by observing a pause of about five seconds between every two words. Furthermore, patients are able to hear and understand words better which have been selected according to their age, education, and range of thoughts, and it is advisable to take these points into consideration not only to expedite the test, but also to avoid blunders. If the test is wrongly conducted, the result will represent the minimal distance, which is clinically quite uninteresting and diagnostically of no value.

These points are of still greater importance if the object of the test is not simply to ascertain the hearing distance, but to demonstrate any improvement after treatment. In the latter case it is important not to use words with which the patient has become familiar from previous examinations.

It is quite possible that at the first examination the established figures are low from the fact that the patient is unfamiliar with the method of testing and becomes confused or excited. It is always advisable, therefore, to follow up the first test by a second one after a lapse of time, when useful figures will be obtained.

The various points above referred to are also brought out in the history of those difficult of hearing; thus, patients understand the language of those they know, especially of relatives, better than that of strangers. The language of women, being more modulated, is better perceived than that of men. Those afflicted with hard hearing may still understand the words of a song in a theatre, but be unable to follow the spoken text, musical sounds being louder and more voluminous.

Hearing Test by Whispering.—The combination field for whispers is usually small (1–1½ feet). The presence of a combination field excludes possibility of simulation (see Simulation).

Hearing Distance for Politzer's Acoumeter.—It is useful to supplement the hearing test for conversational and whispered language by Politzer's acoumeter. This handy and excellent instrument consists of a hard rubber column with a steel cylinder attached, and a small steel lever above the cylinder. The lever may be dropped upon the cylinder at a uniform distance, producing a noise not dissimilar to the ticking of a clock and audible with normal ears at a distance of 15 yards. The result is recorded in a fraction, the numerator of which represents the established distance and the denominator the normal distance (15 yards). The test is commenced in immediate proximity to the ear, with the patient's eyes covered, the distance being gradually increased until the hearing limit has been reached, and beyond. These figures are controlled on the return journey.

II. TUNING-FORK TEST

Testing the ear for the perception of musical sounds is an important link in the system of functional tests.

The tuning-fork should be large, with a handle of at least 8 cm. Handle and prongs should be made of one piece and not be soldered. Nickel-plated forks cannot be recommended, the duration of their vibrations being impaired. For clinical purposes an instrument should be selected free from overtone and with long-lasting vibrations, so that the length of perception may be conveniently compared with that of a normal ear. If the vibrations are rapidly arrested, there may not be time enough to establish precisely the difference of perception between the normal and the pathologic ear. Most of the overtones can be eliminated by weighting the end of the prongs, and in clinical language forks free from overtones are weighted forks. Speaking from a purely technical point of view, there are no forks entirely free from overtones, some of them being always present in the vicinity of the ground tone. Exaggerated weighting of the forks lessens the duration of their vibrations.

Tuning-forks of sufficient size and adequately weighted can be used to test musical scales: the nearer the weights are brought to the base of the fork, the higher will be the sound.

Intensity of Sound.—The intensity of the sound of the tuning-fork is indicated by the distance at which the sound is still perceptible by the normal ear. The sound of low forks, even if firmly struck, is not very intense, while those of a high pitch, even if softly struck, are audible at a considerable distance.

Compass of Sound.—The human ear is capable of perceiving from 12 to about 40,000 (double) sound vibrations. For clinical purposes a fork is used having about 16 vibrations for the lower sound limit (C_2) and 4224 vibrations (c^5) for the upper limit.

Clinical Test Methods.—We distinguish between (*A*) general, (*B*) qualitative, and (*C*) quantitative tuning-fork tests.

A. GENERAL TUNING-FORK TESTS

A long, weighted c^1- or a^1-tuning-fork, or an unweighted a^1-fork, of pure sound, is used for the tests. The kind of fork used should be recorded, as a guide for possible control tests.

(1) **Weber's Test.**—The sounding fork is placed against the vertex of the patient, who will have to state where he hears the sound. If both ears are normal or equally affected, the sound will be heard either in both ears, in the head, at the point of contact, or in space. In unilateral aural affections there will be lateralization of the sound to one side. If the sound-conducting apparatus (of the external and middle ear) is diseased, the sound will be perceived on the pathological side,—*i.e.*, lateralized to that side. If the sound-perceiving apparatus (of the internal ear: labyrinth, auditory nerve) is diseased, lateralization ensues toward the healthy or less affected side. If lateralization is not sufficiently distinct, the patient will be unable to make a definite statement. In this case, lateralization can be intensified by using a lower-pitched fork or by selecting another point of contact in the cranial medial line (forehead, teeth, nape of the neck, occiput).

The test is explained as follows: Lateralization to the healthy or less affected side is easily intelligible in cases of a diseased perceiving apparatus. If the auditory nerve on one side is intact, that of the other diseased (atrophied, for instance), and the labyrinth degenerated, the sound of the fork will, as a matter of course, be lateralized to the normal or less affected side. Lateralization in cases of an affected conducting apparatus may be theoretically explained as follows: The sound of the fork on the vertex is perceived through the cranio-tympanic and cranio-labyrinthine conduction. An affection of the conducting apparatus will not affect the cranio-labyrinthine conduction. On the other hand, the sound waves reaching the bone by the route of the cranio-tympanic conduction are normally changed into air waves in the middle ear, and it is only after this transmutation has taken place that they will reach Corti's organ. This transmutation of waves in the middle ear causes some loss of intensity, and, besides, a certain quantity of sound waves passes out through the external auditory meatus. This fact is easily demonstrable by the otoscope: if the patient's ear is connected with that of the operator, the latter is able to hear the sound of the fork. But if

there is an obstacle interfering with sound-conduction in the external canal or middle ear, the normal loss in intensity is either completely eliminated or considerably reduced, so that the fork will be heard more intensely than in normal conditions. The objective prolongation of the cranial conduction in affections of the sound-conducting apparatus follows from the same considerations.

Weber's test is of clinical value only in so far as lateralization unquestionably indicates an aural affection, as in bilateral normal conditions it cannot possibly occur. It is of considerably less value for differentiating between the affections of the sound-conductors and those of the sound-perceivers. In affections which have both otoscopically and functionally been proved to be such of the sound-conductors, the labyrinth being perfectly intact, some patients will lateralize to the healthy side, being under the impression that an otherwise inferior, affected ear would not be able to perceive the sound of the fork better than the good ear. Thus, they consider it but natural that the healthy ear should perceive the sound in Weber's test better than the affected one. These patients are not sufficiently mature to answer questions correctly. Again, in positively demonstrated affections of complete destruction of the labyrinth, the sound coming from the fork may be lateralized to the affected side, although the reverse would have been expected. The explanation is that, although the affected ear no longer exercises any physiological function, especially after the radical operation, it yet acts as a powerful mechanical resonator, and the sound is thus erroneously taken to come from the resonator. This is a regular occurrence in experiments upon the normal ear with artificial occlusion of the external meatus.

(2) **Schwabach's Test.**—The vibrating fork is placed upon the middle of the forehead, the patient being requested to state whether or not he hears the sound of the fork. To obviate fatigue or suggestion, the fork is removed and replaced at short intervals, the patient having to answer the question on each occasion. If there is any doubt as to whether the patient can distinguish between the mere vibrations and the sound they produce, the fork is placed against the wrist or patella: if the patient really perceived the sound from the middle of the forehead, he will now state that he can only perceive vibration at the wrist or patella; if, on the other hand, he had only perceived vibrations at the forehead and erroneously taken them for sounds, he will commit the same mistake with the fork against the wrist or patella.

The duration of sound-perception is divided into normal, shortened, and lengthened, and their various degrees can be best established by the Kittlitz tuning-fork, as recommended by Bloch, or by the same instrument as modified by Bernd, or by Gradenigo's fork.

In affections of the sound-conductors, the cranial conduction is prolonged; in those of the middle ear it is shortened. The degree of shortening practically corresponds to the degree of deafness, while the degree of prolongation does not correspond to the degree of the affection. In acute otitis media it will usually be found greater than in chronic cases. The physiologic explanation of Schwabach's test is the same as that of Weber's test.

It is an interesting and fundamental fact in Schwabach's test that the affected side has a preponderating influence in the middle of the forehead. If, in a unilateral affection, the cranial conduction of that side is prolonged, it will also be prolonged from the middle of the forehead, but shortened in unilateral affections of the middle ear. Some authors apply Schwabach's test from the mastoid processes instead of the forehead, erroneously supposing that the conduction thus found represents a picture of the cranial conduction of a particular side, which, however, is not the case. For instance, the conduction found at the right mastoid will furnish a picture of cranial conduction representing the capacity of the right ear to a greater extent and that of the left ear to a less extent, but the degree of participation of either ear cannot be precisely determined.

In my opinion it is preferable, therefore, to provide topographically equal conditions for both cranial halves by placing the fork in the middle of the forehead.

The duration test in normal individuals sometimes discloses considerable individual deviations. Abnormal duration, however, can only justify the assumption of an aural affection if the other functional tests, especially the determination of the aural acuity, should confirm it.

Schwabach's test is a very valuable diagnostic aid if practised with the necessary precaution.

(3) **Rinne's Test.**—This test results from a comparison of the duration of the cranial and air conductions of the same side. If the sound of the fork is no longer perceived at the mastoid, a normal ear can still perceive it for some time through air conduction (Figs. 48, 1), the duration being normally longer through air than through cranial conduction. Rinné's test is called positive if the air conduction preponderates over cranial conduction, and negative in the reverse case. In aural affections the air conduction is shortened, and the fact of its being shortened indicates the presence of an aural affection, whatever its origin may be. Cranial conduction, however, varies, and, as has been shown in the description of the Weber and Schwabach tests, is prolonged in affections of the sound-conductors and shortened in those of the sound-perceivers, as compared to normal.

This leads to the following divisions of Rinné's test (Fig. 48, 1–7):

1. In the normal ear, Rinné's test is positive with normal duration of perception through either cranial or air conduction (Fig. 48, 1).

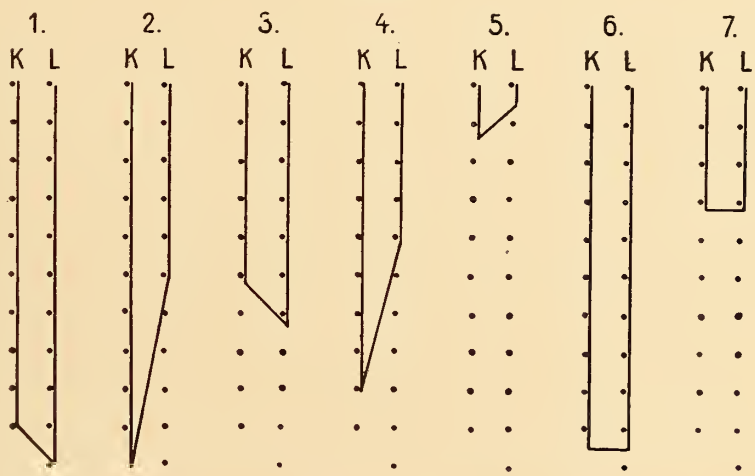
2. In affections of the sound-conducting apparatus, Rinné's test is negative, with shortened air conduction and prolonged cranial conduction (Fig. 48, 2).

3. In affections of the sound-perceiving apparatus, Rinné's test is positive, with shortened cranial and shortened air conduction (Fig. 48, 3).

4. In affections of the sound-conducting and sound-perceiving apparatus, the Rinné test is negative, with shortened cranial conduction (Fig. 48, 4).

5. In advanced stages of hearing insufficiency, the Rinné test is always negative (Fig. 48, 5), whether the affection involves the sound-

Fig. 48.



Graphic representation of Rinné's test for clinico-diagnostic purposes. *K*, cranial conduction; *L*, air conduction.

perceiving or sound-conducting apparatus, or both. In the most advanced stages of progressive insufficiency, air conduction always diminishes more rapidly than cranial conduction. In very advanced cases, therefore, Rinné's test is of no diagnostic value.

The test is indifferent, undecided, or simultaneously positive and negative:

6. In light affections of the sound-conductors, in which there is a prolongation of the cranial conduction and a slighter shortening of the air conduction in such proportion that the same duration results from both ways of conduction (Fig. 48, 6).

7. In many advanced cases of middle-ear affections (Fig. 48, 7).

Rinné's test is an exceedingly valuable diagnostic aid, provided the

precautions resulting from a study of Fig. 48 are observed (comparative measurement of the time of perception).

(4) **Politzer's Test.**—While the patient holds a swallow of water in his mouth, the tuning-fork is held before his nose. The sound will be perceived stronger during the act of swallowing if the tube is normal, the volume of tone suddenly increasing. The test proves the importance of the pharyngo-tympanic air conduction.

The test is negative in tubal affections. In unilateral affections, the sound will only be heard through that tube which opens during the act of swallowing.

Thus, the condition of the Eustachian tube can be rapidly established by this test, which is particularly valuable in cases where other methods of tubal examination are not available.

(5) **Gelle's Test.**—This test is carried out with a weighted C^1-fork and a rubber tube provided with an olive nozzle and a rubber bulb. The latter has an aperture at the side, which can be closed with the thumb (Bloch). The moistened nozzle is fixed air-tight in the auditory meatus and the sounding fork placed upon the bulb. On compressing the latter, with its lateral aperture closed, the perception of the sound will be considerably diminished. On liberating the aperture and thus reducing the increased pressure to normal, the intensity of the sound increases to its former level.

The following theoretical considerations will explain the test:

Compression of the bulb serves to raise the air pressure in the area of the sound-perceiving apparatus down to the labyrinth, causing the mobile parts of the labyrinthine window (plate of stapes, membrane of the cochlear window) to protrude into the labyrinthine spaces. This leads to an increase of the intralabyrinthine pressure, with diminution or arrest of mobility (vibration) of the stapes and the membrane of the cochlear window. This condition amounts to an artificially produced disease of the labyrinth, in which sounds are perceived less clearly than under normal conditions. On restoring the original condition by releasing the aperture of the bulb, the labyrinth returns to its normal condition, and the sound is perceived more strongly.

The normal result of Gellé's test is called positive. It is negative if the mobility of the parts in the area of the labyrinthine window is arrested by congenital or acquired changes,—*i.e.*, experimental changes of the air pressure will not alter the perception of the sound from the fork. In cases of diminished mobility the test is indistinct; sometimes it is at first negative but becomes positive on repeated application, evidently in consequence of the massage of the ossicles affected by applying the test.

It is an important fact that, under normal conditions of the osseous capsule of the labyrinth, the experimental elevation of the air pressure leads only to functional disturbance in the acoustic labyrinth (cochlea), while the semicircular canals and vestibule remain unaffected.

When placing the fork against the mastoid in this test, the cranial conduction will be shortened in proportion to the increase in air pressure. In Gellé's test the fork may also be placed upon the vertex in cases which do not lateralize the sound in Weber's test. At first, the patient lateralizes the sound to the side blocked up by the nozzle, the latter being an impediment to sound-conduction. An artificial affection of the labyrinth produced by air compression will cause the sound to be lateralized to the other side.

Gellé's test is exceedingly valuable, furnishing as it does a reliable picture of the condition of the labyrinthine window. The negative results have come to be considered a special diagnostic sign of otosclerosis, and sometimes render an early diagnosis of this serious affection possible.

The positive vestibular reaction in Gellé's test is considered to be a symptom of fistula (see p. 91).

B. QUALITATIVE TEST BY TUNING-FORK

The qualitative test is made by tuning-forks of different pitch or, in certain cases mentioned below, by a continuous series of differently pitched forks.

In testing for quality, or timbre, air conduction is availed of. The simplest test is made with a weighted deep (C^1) and an unweighted high (c^4) tuning-fork.

Perception of the deep sound is considerably diminished, or inhibited, by an obstacle to conduction. Shortened perception of a high sound points to an affection of the cochlea, which, according to Helmholtz's theory, is situated in the vestibular section reserved for the perception of high sounds. This brings us to the following conditions:

1. If there is an obstacle to conduction, C^1 is negative or considerably shortened c^4 normal.

2. If there is an affection of the sound-perceivers in the area of the vestibular section of the cochlea (the perceptive place for high sounds), C^1 will have normal, c^4 shortened duration. This simple test with a deep and a high fork enables us to diagnose any obstacle to conduction. But an affection of the perceiving apparatus can only be diagnosed with it if the vestibular part of the cochlea is affected and the remaining parts of the cochlea are healthy. Nearly all cochlear affections emanating from a middle-ear affection correspond to this relation, and the test with the unweighted c^4-fork is exceedingly valuable in

determining whether in otitis media the cochlea is still intact or already involved. In the former case, the c^4-fork will be heard through air conduction either at normal time or shortened only a few seconds. The first sign showing that otitis media has spread to the labyrinth consists in considerably shortened perception of c^4-fork through air conduction.

This unweighted fork is selected for special reasons: Its pitch is high enough to make a diagnosis of the condition of the vestibular part of the cochlea, while the duration of its vibrations is still long enough to admit of convenient comparison between the time of perception of the affected with that of the normal ear and to establish the exact degree of shortening in seconds. A good c^4-fork should vibrate for 54–60 seconds, while even the best c^5-fork will not vibrate for more than 8–12 seconds, which is not long enough to admit of a convenient and exact comparison of the time of perception between a diseased and a normal ear.

It will thus be seen that the forks C^1 and c^4 will not meet the requirements of all cases. Where they fail, it will be necessary to use a system of serially pitched forks. For this purpose the following instruments have been devised: (1) the serial fork-pipe instrument of Bezold-Edelmann, which is provided with resonators; (2) Stern's sound variator; (3) the harmonica of Urbantschitsch.

Bezold-Edelmann's continuous-sound series consists of 10 weighted tuning-forks, each of which can be varied in pitch by shifting the weights. The totality of the 10 forks contains every sound from C^2 to a^2. There are three pipes for the higher sounds of the scale commencing with a^2, the compass of the large pipe extending from a^2 to a^3, that of the small one from a^3 to a^4. The compass of the Galton pipe extends from a^4 to the highest note limit.

Recently the monochord has been recommended to determine the highest tone limit. It contains four additional unweighted forks, so as not to do without any forks entirely in a pipe system. The vibrations of these forks are as follows: g^3 1550, c^4 2069, g^4 3100, c^5 4138.

In Stern's sound variator an almost imperceptible transition from one sound to another may be accomplished by changing the length of the pipes. The clinical use of this excellent instrument, however, is handicapped by the necessity of using bellows, making it untransportable, and also by its cost.

With the harmonica of Urbantschitsch sounds of uniform strength are attainable by attaching pipes of various lengths to a harmonica. This instrument is particularly suitable for determining the remaining degree of hearing in very advanced cases and for the examination of deaf-mutes.

The routine use of a continuous series of sounds is not feasible, owing to the length of time required for examination, amounting as it does to one or two hours. It will, therefore, be reserved for cases in which the ordinary methods are not sufficient. In connection with these points, the following remarks may be made:

(1) It has already been pointed out that the relation of "C^1 positive and normal, c^4 shortened and negative," only holds good for certain forms of affection of the cochlea, particularly for those which have remained confined to its vestibular part. If, on the other hand, the pathologic changes permeate the cochlea or the auditory nerve in the shape of foci, the hearing ability will be suddenly and irregularly impaired or destroyed for certain series of sounds and remain normal for others. This results in the so-called acoustic or sound islands (Bezold), and it stands to reason that these can only be localized by tests with a continuous series of sounds. The diagnosis in many cases of traumatic affection of the labyrinth can only be made after a test with one of these instruments. So far as forensically important cases are concerned, it will therefore be advisable to make a test with a continuous series of sounds the basis of functional examination.

(2) An important part in the symptomatology of subcortical sensory aphasia is played by word-deafness. Patients thus afflicted at first convey the impression of being totally deaf, being entirely unable to understand words, so that all conversation has to be carried on in writing. Music, however, is perceived by them. In these patients, the question of sound-perception can only be determined by the continuous acoustic method, and it is only due to these tests that it is known to-day that patients with subcortical sensory aphasia may have a good and sometimes perfectly normal ear for music.

(3) The examination of deaf-mute children was originally restricted to the quantitative measurement of acoustic remnants. It was on Itard's suggestion that deaf-mutism in its wider sense was divided into five groups or degrees. These groups comprise all those children whose sense of hearing has been impaired to such an extent as to disqualify them from following instruction at school. These children, if left to themselves, develop their own way of making themselves understood by gestures or signs, but articulate language is usually denied them.

The five groups are the following: (*a*) total deafness, (*b*) ability to perceive great noises, (*c*) ability to hear vowel sounds sung with a loud voice, (*d*) ability to hear easy words spoken with a loud voice, (*e*) ability to understand ordinary conversational language at a distance of 1–2 m.

The deaf-mute inmates of an institution comprise a mixture of these various degrees. Instruction, and methods of imparting it, must

be adapted to average preparatory conditions. If all the children of whatever degree of deaf-mutism are brought together in one class, those with a partial sense of hearing will learn their lessons better and quicker than those totally deaf. The fact is that for groups *b* and *c* the quantitative and qualitative tests are not sufficient to allow of an opinion as to their possibilities, and it is highly questionable whether in individual cases the acoustic remnants benefit the children in instruction and education or not. (There is no need to emphasize specially the great importance of useful acoustic remnants in the development of intelligence and knowledge in school children. The chief aim in the education of deaf-mute children is to impart to them a sound-language and articulation. The attainment of these objects is powerfully advanced and facilitated by the smallest useful remnants of hearing. The methods of instruction can avail themselves with the greatest benefit of any such remnants that may be preserved (Bezold, Urbantschitsch), and, as an immediate consequence of this fact, attention should be centred in a system according to which children with useful remnants of hearing are placed in a special class. Instruction in such a class may be much more rapid than in classes reserved for the totally deaf and those with useless remnants.)

The quantitative tests for groups *b* and *c* are not sufficient to establish the value of the remaining acoustic remnants; this can only be accomplished by the qualitative test with Bezold's continuous series of sounds, since stroboscopic examinations of the language have shown that a compass from b^1 to g^2 (Bezold) must at least be preserved for hearing the spoken language.

It is evident that only such acoustic remnants can be serviceable which lie within that area, and to determine upon the value of these remnants the continuous-sound test is indispensable. The test is in all cases monaural. The ear not-under examination is firmly plugged with cotton saturated with glycerine. It is advisable to commence the test with the c^1-fork, proceeding downward first to the lowest end of the series and establishing the lowest sound limit. Only then should the upper series be dealt with in the same way, until the upper sound limit has been reached. The patient should be carefully watched during examination, and, if there should be any fatigue apparent, the examination should be interrupted for some time, if necessary for several hours.

This test presupposes the ability on the part of the child to display a high degree of attention and concentration, qualifications which are only acquired at school. For this reason no test should be made before the end of the first school year. In less intelligent children no reliable test can be made before the end of the second, third, or fourth year.

C. QUANTITATIVE TEST BY THE TUNING-FORK

1. **Measurement of the Difference of Perception.**—The object of the quantitative test is to establish the difference in time between the duration of perception in the affected and a normal ear. Generally speaking, any increase in this difference is proportionate to the degree of hearing insufficiency present. As a matter of course, none but air conduction is of any particular importance for this measurement.

As soon as the patient indicates by raising his hand that he no longer perceives the sound of the fork a stop-watch is set going and the moment the sound is no longer heard by a normal ear the watch is stopped. This method, however, is very lengthy and applicable only if the examiner himself is of normal hearing.

2. **Quantitative Test with the Bezold-Edelmann Graded Sound Series.**—A comparatively simple and sufficiently exact test can be made with Edelmann's tuning-forks, which are of excellent workmanship. Every experienced examiner becomes accustomed to a certain intensity of stroke, whether the large forks are struck with the palm of the hand, with the thumb, or, as is usually done with small forks, with a striker. Every examiner can determine for his own instrument the time of perception, provided, of course, the stroke is always the same. The degree of shortened perception in the patient can then be numerically determined without difficulty, and the graphic record of the result represents "Bezold's acoustic relief." Its working basis for deep and medium forks may be formed by the duration of vibrations when the forks are unweighted (Bezold) or when weighted (Fig. 51).

3. **The Gradenigo Forks.**—A black triangle, divided into four parts by lateral lines, is attached to the weights of a tuning-fork. The fork being struck, two gray-tinted triangles become visible to the eye, which in the course of the vibrations cover each other at a progressive rate. The time required from the partial covering of the triangles at a definite division line to the complete stand-still of the fork is noted. This would show, for example, that the large C^1-fork has sounded for 72 seconds from the moment of covering at the lowest division line until the end. Using the fork at a clinical examination, it is allowed to sound until the triangles are covered at the established division line; at that moment the fork is brought to the patient's ear and note is taken of how long he can perceive the sound. Knowing that a normal ear can perceive the sound for 72 seconds, counting from the established division line, it is very easy to determine the degree of shortened perception without its being necessary to compare again the time of perception with that of the normal ear at the time of examination.

4. **The Kittlitz-Bernd Fork.**—The instrument devised by Kittlitz at Bloch's instigation may be used for forks of different pitch, but is

best suited for the unweighted c^1-fork of Edelmann. It is easier to read off the result from this device than from that of Gradenigo. Latterly the Kittlitz fork has been considerably improved upon by Bernd.

In spite of a number of experiments, it is impossible to express the hearing acuity in exact percentage, it being necessary to content one's self with stating the degree of shortening in seconds and the approximate percentage. In many cases it is sufficient to distinguish between four degrees of pathological perception of the tuning-fork, as follows: (*a*) slight shortening, (*b*) medium or moderate shortening, (*c*) considerable shortening, (*d*) no perception (sound-deafness).

III. TEST OF CRANIAL-BONE CONDUCTION BY NOISES

(1) *Watch-test.*—The ticking of a watch is distinctly heard by the normal ear from any part of the cranial bones up to the age of 40, and at least from the mastoid up to the age of 50. Suppressed perception of the ticking through the cranial bones points to an affection of the sound-perceiving apparatus. Although the watch-test is not of very great practical importance, it is nevertheless an appreciable adjunct in connection with the other functional tests. Affection of the sound-perceiving apparatus can, however, not be diagnosed therefrom unless there are other symptoms pointing to an involvement of the inner ear. Younger men, with a perfectly normal sense of hearing, may in exceptional cases fail to perceive the ticking of a watch through the cranial bones, while in children the test may be negative from want of attention. Neurasthenics are sometimes under the impression of hearing the ticking, but control with a stop-watch will immediately disclose the real state of things, since the patient will still believe he can hear the watch ticking after it has been stopped.

Testing for distance by means of a watch through air conduction is of no practical importance.

(2) *Testing with Politzer's Acoumeter.*—The acoumeter represents a very simple means of establishing the degree of hearing acuity so far as noises are concerned (see p. 67). It is of less importance for testing cranial conduction, seeing that the comparatively loud noise produced by the acoumeter would only be inaudible in considerably advanced stages of affection of the inner ear, and patients frequently experience difficulty in separating the tactile from the hearing sense when the apparatus is in motion.

IV. DEMONSTRATION OF UNILATERAL DEAFNESS

It was pointed out in the section on the Physiology of the Ear that it is impossible to isolate completely the function of one ear to the exclusion of the other, either by way of air or cranial conduction. Even

when the external meatus is closed in the most laborious manner by filling it with glycerine and plugging it with cotton, the sound-conduction to that ear will not be completely suppressed. Besides, in the functional test of one ear there is no way of preventing the other ear from participating in the perception of sound by way of cranial conduction.

We are, therefore, content in the monaural test to have the meatus of the other side closed with a moistened finger tip, although this will not prevent the examined person from correctly repeating words spoken in conversational language at a distance of from 6 to 10 yards. This can be easily demonstrated in a normally hearing individual by plugging both ears.

If in a case of unquestionable unilateral total deafness the normal ear is plugged as described, the deaf ear will have an apparent acuity of 1–2 yards for conversational language. Accordingly, we arrive at the following rule: If, on examination, we find an acuity of 2 yards C., or less, in one ear, it will be necessary to test this ear by itself, in order to ascertain whether this is the actual hearing distance or whether the ear is totally deaf and the apparent hearing distance is only the result of functional coöperation of the other side which cannot be entirely excluded.

The following methods serve to establish unilateral deafness:

1. **Testing with the Hearing Tube.**—When speaking the test words through a long hearing tube into the ear to be examined, cranial conduction to the other ear will be of less importance than in the ordinary arrangement, as only easy and isolated words can be perceived by the opposite ear, which is closed with a finger. I have used for a number of years a rubber tube 4 meters long and 1.5 cm. clear diameter, fitted with a metal olive and a hard rubber funnel. If conversational language and whispering, or conversation alone, are still distinctly heard, and if the test words are promptly and correctly repeated, it shows that the examined ear has still a positive sense of hearing for language. Should only some of the words be understood, or none at all, or should there be any hesitation or mistakes, a diagnosis of deafness for spoken sounds is justified.

2. **Test with the Unweighted a^1-Fork. Bezold's Test.**—This test was recommended by Bezold as a simple means of establishing unilateral deafness for spoken sounds. Bezold-Edelmann's a^1-fork is best suited for this test. Its pitch is high enough to be used as a test for the cochlea, and yet deep enough to be heard by the other well-plugged ear by way of air conduction. As a matter of course, this test is only carried out for air conduction.

The value of this method consists in the following: Perception of the a^1-fork, even though considerably shortened, is a conclusive proof of functional ability of the tested ear. Complete absence of perception for air conduction in this test can only be regarded as a proof of deafness

if the result of the other tests points to unilateral deafness. For clinical purposes the a^1-test should preferably be combined with Stenger's test (see below).

3. **Stenger's test** requires two tuning-forks of precisely identical pitch. The test for the normal ear is conducted in the following way: One of the forks is held in front of the healthy ear at a distance of 6–8 cm., the patient, of course, stating that he perceives the sound on that side. Upon bringing the second fork, while sounding, near the other ear at a smaller distance, say 4–1 cm., while the position of the first fork remains unchanged, the sound will only be heard by the ear which is nearer a fork. On removing the latter, the original perception is restored. It is possible, therefore, to suppress the perception of the first ear by closer approach of an identical fork to the meatus of the other ear. If it is desired, for instance, to test for right-sided deafness, one of the forks is held at a distance of 6–8 cm. from the left meatus; the second fork is then brought as near as possible to the meatus of the right ear. Should the latter possess functional hearing ability, the perception of the left side would be suppressed as the right fork is being approached, and the patient will state that he can hear the sound only with his right ear. On removing the fork from the right ear, perception of the left ear immediately reappears. Should, however, the right ear be actually deaf, even the closest approach of the second fork to the right ear will remain without effect, and the patient will state that he has heard the sound from the left side without interruption.

It is advisable in this as well as in all other tuning-fork tests that the examiner stands erect behind the patient, who sits down, in order to avoid error and confusion. Nor should the patient be allowed to know that two forks are being employed.

Stenger's test is not only interesting theoretically, but is also of clinical value in that the method of questioning is uncommonly simple, the patient having merely to state whether he hears the sound on the right or left side. The difficulty patients experience in stating whether or not they still perceive a sound (as in the tuning-fork tests), or in indicating the precise moment when they cease to perceive a vanishing sound, is entirely done away with. Stenger's test is very valuable and indispensable for the demonstration of unilateral deafness, but the two forks used should be of exactly identical pitch. Personally, I always use Bezold-Edelmann's unweighted a^1-forks and combine the Stenger and Bezold tests.

Complete uniformity of two tuning-forks is established in the following way: Strike one of the forks and approach with it the silent one with prongs parallel, and the second fork will commence to covibrate if of identical pitch.

4. **Alarm Instruments.**—Bárány has devised an alarm drum, Neumann a galvanic alarm instrument, Voss a water jet bellows, for the purpose of creating a great noise with the intention of excluding the physiological coöperation of the healthy ear when testing for deafness of the other one.

V. DEMONSTRATION OF SIMULATED HEARING INSUFFICIENCY, SIMULATED UNILATERAL AND BILATERAL DEAFNESS

Simulated hearing insufficiency with normal function of the ear is recognized by the fact that the result of the functional test does not agree with that of the test for distance. The tuning-fork test will often be perfectly normal, so that there is no basis whatever in the clinical functional findings for the alleged hardness of hearing. Another important sign is the complete absence of a combination field, and the method in which the individual repeats the test words. The patient's eyes being bandaged, he will make contradictory statements by professing inability to understand words spoken close by, while others, spoken at a greater distance, are perceived. There is hesitancy in repeating, although the words are spoken within positive hearing distance, the object being to create the impression that there was difficulty in catching them. He innervates the facial and appears to listen with the closest attention. He requests words to be repeated, and responds by repeating at first nothing but the article, then the first syllable, and so on, until at last he repeats the entire word. A genuine patient never acts in this way: he will combine when hearing indistinctly, but he will never be able to piece words together syllable by syllable.

It is a more difficult task to recognize aggravation. This implies that a person hard of hearing simulates a higher degree of insufficiency than really exists. It may be a matter of great difficulty to establish the true degree of hearing insufficiency, especially if the patient has been examined before on several occasions and is careful enough not to overdo his exaggerations. However, experience on the part of the examiner, repeated examination with bandaged eyes and on different days, will usually disclose the facts.

Exposure of simulated unilateral deafness does not present any serious difficulties. If the other ear is normal, suspicion will be aroused by the patient pretending not to hear words spoken closely to the supposedly deaf ear in the monaural test, knowing as we do that in total unilateral deafness and normal hearing on the opposite side (which is closed with a moistened finger) there is still an apparent hearing distance of at least 1 yard C. Another aid is Stenger's test, which is based on the fact (first discovered by Politzer) that, with equal quality, the weaker sound-perception is eliminated by the stronger.

The test in such a case is made as follows: Two a^1- or c^1-forks are used. One is held at a distance of 6–8 cm. before the normal ear, after which the other one is gradually approached toward the supposedly deaf ear at the closest possible range. The patient, with bandaged eyes, the examiner standing behind him, is requested to state on which side he perceives the sound. If the ear is really deaf, the sound will, of course, be perceived at the sound side; if, however, deafness is simulated, the closer approach of the fork toward the supposedly deaf ear will have suppressed the perception on the first side. The individual positively perceives the sound by the supposedly deaf ear, but, since he feigns deafness in that ear, will pretend not to hear any sound at all.

Further valuable assistance is given by a survey over the entire system of functional tests:

In unilateral deafness the results of the tuning-fork tests, hearing distance, etc., are very characteristic: the findings will always disclose an affection of the conducting and perceiving apparatus, or of the latter alone, since an affection limited to the sound-conducting apparatus can never cause deafness. In simulation the statement of functional facts is full of contradictions, and on going into details of the results of the functional tests it will not be difficult to narrow them down so as to form a basis for the diagnosis of simulation.

The simulation of bilateral deafness or deaf-mutism may cause considerable diagnostic difficulties.

It is important to observe the behavior of the patient. A genuinely deaf person will anxiously look about, closely watch our lips during conversation in order to read the words therefrom. These patients are elated at the least ray of hope we give them and could be easily persuaded to undergo treatment.

The simulator,, on the other hand, looks apathetic or recalcitrant, especially if he has already undergone several examinations and believes that he has had the best of it. He stands with downcast eyes, avoiding to look into your face. From the first we are imperiously informed that only written conversation can be carried on, and a formidable array of note-books are produced to substantiate the statement. He requires neither treatment nor improvement. He has no time for treatment, as he is "just off for a trip" or he "does not live in town": all he requires is a certificate stating that he is stone-deaf. Young persons are usually exposed in a short time, and, on getting excited, they fling writing to the winds and react to conversation.

At the same time, attempts to force a sudden exposure are not advisable. In making the remark, "Very well, I can see you are really deaf, you can go," we may involuntarily accompany our words by move-

ments or gestures which are correctly interpreted by a genuinely deaf person, and the fact of his responding to them is no proof of simulation.

In difficult cases it will be necessary to keep the patient under observation for some time when he thinks himself unobserved, in the street, at home, etc.

The authorities are also a reliable source of information. I am in the habit of investigating patients' personal affairs at their place of birth, sending a question blank to the local authorities with a request to fill in all valuable data on school attendance, communications of teachers, employers, etc.

In apathetic subjects who are supposed to have acquired deafness at a later period, and in those where there is a combination of alleged deafness and an affection of the brain, notably idiocy, it may be difficult or impossible to decide whether there is deafness or simulation. I have been completely at a loss to make up my mind in a few cases of idiocy which had not reacted to any noise whatever for a number of years as to the genuineness of the aural affection.

There is a difference between diagnosing aggravation or simulation and convincing the accused of the fraud. The diagnosis is positive in many cases on the basis of functional findings, and yet the person in question may not be willing to admit that he is shamming. At the same time, the absence of admission is of no consequence from a diagnostic point of view, and personally I do not care whether I obtain an admission or not.

The patient should under any circumstances be very civilly treated during the examinations. It would be wrong to create the impression that his statements are distrusted or that there is an attempt to expose him. If simulators find their position untenable, they are apt to leave brusquely and decline any further examination, thus rendering the completion of a logical diagnostic structure impossible. As soon, however, as we are able to make the diagnosis on the basis of the facts we have elucidated, we may try to persuade the simulator to admit his false position. This is best accomplished by a kindly talk. We explain to him that we have established the positive fact that he is shamming, adding that we will now repeat the functional test, to which he is requested to pay the closest possible attention. Such an appeal is generally successful.

Furthermore, the suspicion of simulation should not be too readily entertained. I always give supposed simulators the benefit of the doubt, unless there is direct proof to the contrary, furnished by contradictory statements in the functional test. Even when confronted with some irregularity in the results, allowance should be made in children for becoming fatigued and in adults for neurosis or hysteria. As to methods applicable in exposing frauds, I refer to Politzer's text-book and Hammerschlag's "Compilations."

VI. FUNCTIONAL EXAMINATION OF THE SEMICIRCULAR CANALS AND THE VESTIBULAR APPARATUS

1. LABYRINTHINE NYSTAGMUS AND THE METHODS OF ITS OBSERVATION

In the functional examination of the static labyrinth it is necessary to establish the labyrinthine reflex excitability and to observe the presence or absence of labyrinthine manifestations of excitability.

In order to find out whether nystagmus is present, one of the eyelids is slightly raised, and patient is requested to look at an object 1–2 yards distant, first in a straight line, then toward the right and left. Spontaneous nystagmus, if present, is bilateral in the majority of cases and of equal character in both eyes. Horizontal nystagmus is diminished by increased convergence, while nystagmus of less intensity can hereby be completely suppressed. In labyrinthine nystagmus the rhythmic movements of the bulb are composed of an intense movement toward one side and a less intense slow movement toward the other side, and the nystagmus is designated by the direction of the more pronounced movement. Hence the expressions "nystagmus to the right" and "nystagmus to the left."

In regard to the movements of the bulb in straight nystagmus, in which the bulb is displaced in a straight line, we distinguish horizontal, oblique, and vertical nystagmus. If the bulb rotates, we designate the nystagmus as rotatory. In many cases the entire movement is composed of a rotatory and a horizontal contingent. This is called horizonto-rotatory nystagmus, or rotatory nystagmus with a horizontal component, or horizontal nystagmus with a rotatory component, according to which component preponderates, if any.

According to the extent of the twitchings, we distinguish between fine and coarse, rapid and slow nystagmus. Exact figures, if desired, must be established by counting per minute. The intensity of the nystagmus is established by the relation existing between nystagmus and direction of vision. Nystagmus of slight intensity is only demonstrable with vision toward the same side; for instance, nystagmus of slight intensity to the right can only be demonstrated with vision to the right (first degree of intensity). The second, or medial degree of intensity, is present if there is nystagmus with vision toward the same side and with straight vision (looking at an object several yards distant or without fixation behind Abels's spectacles). In the third and highest degree of intensity, nystagmus is entirely independent of the direction of vision and is demonstrable in any position of the bulb, including vision toward the opposite side. Thus, nystagmus to the left will not be inhibited by vision to the right.

2. SPONTANEOUS LABYRINTHINE NYSTAGMUS

Pathological nystagmus, which occurs in certain affections, is also called spontaneous nystagmus, of which we distinguish three forms: (1) In many affections of the labyrinth, nystagmus to the right occurs upon vision to the right, and nystagmus to the left upon vision to the left. This is designated nystagmus toward both sides, or nystagmus in the end-positions of the bulb. (2) Nystagmus toward the affected side is recognized by the twitchings occurring toward the affected aural side. (3) If there is pronounced nystagmus toward the opposite (healthy) aural side, we so designate it.

Spontaneous nystagmus toward both sides is always of slight intensity. It never exceeds the first degree above referred to, while the other two forms of spontaneous nystagmus may reach any degree of intensity. It is advisable, therefore, in all cases first to test for spontaneous nystagmus with lateral vision, and then to establish the behavior of vision to the right, straight, and to the left. In normal individuals there is no spontaneous labyrinthine nystagmus. Nor is it demonstrable in many pathological cases where the semicircular canals on both sides have been destroyed a considerable time ago. In all other cases the pathological condition of the semicircular canals is demonstrated by the presence of spontaneous nystagmus.

In regard to the relation of the spontaneous nystagmus to the other labyrinthine symptoms, to the reflex excitability of the labyrinth and hearing acuity, the reader is referred to the chapter on inflammatory affections of the labyrinth.

3. LABYRINTHINE NYSTAGMUS AND VERTIGO AS DIFFERENTIATED FROM THE OTHER FORMS OF NYSTAGMUS AND VERTIGO

Labyrinthine nystagmus is characterized by the fact that the rhythmical movements of both eyes occur simultaneously and that the forward (first) movement is always more intense than the backward (second) movement. This fact will enable even the less experienced easily to recognize and describe a labyrinthine nystagmus from its direction. If the labyrinthine nystagmus is of slight intensity, it is only demonstrable with vision toward the same side, while none but high degrees of intensity can be demonstrated with straight vision. The highest degree of intensity is demonstrated by the fact of nystagmus being independent of the direction of vision, so that it can be distinctly observed in any position of the bulb. In slight degrees of intensity, labyrinthine nystagmus is only occasionally visible, notably at the time of vertigo, or it may be entirely absent. If, finally, in spontaneous labyrinthine nystagmus, vision is repeatedly directed toward the same side, alternately to the right and left, there will usually be

rapid diminution of the nystagmus, sometimes entire suppression of it for a time.

Congenital nystagmus can be easily distinguished from the labyrinthine form. It is composed of oscillating or undulating movements, there being no particular intensity toward any side. Congenital nystagmus is usually coarse and of high intensity, so that it can often be distinctly observed at a distance of several yards. It is not associated with vertigo.

Optical nystagmus has the following characteristics as compared to the labyrinthine form: It can be traced to definite ocular affections (affections of the orbital muscles, anomalies of bulbar development, anomalies of refraction), provided it is caused by a pathological factor situated within the orbit itself. Optical nystagmus, caused by fixed contemplation of objects in motion, disappears on closing the lids or by simply discontinuing fixation temporarily or entirely.

Neurotic or neurasthenic nystagmus occurs only with extreme lateral vision and is considerably increased by repeated intense vision toward the right and left.

Labyrinthine vertigo occurs by subjective, illusory oscillations, which are more frequently perceived as rotations of the surroundings than of the patient's own body. In the latter case the direction of rotation usually corresponds to that of the nystagmus; in the former case rotation appears to be in the opposite direction. The details of an attack of vertigo will be remembered by the patient more clearly if the attack was severe and continuous; otherwise the rotatory movements will not be retained in the memory as a characteristic occurrence or no notice may be taken of them at all. If the attack was severe, not only the fact of the oscillations but also their direction will be remembered. The principal deuteropathic manifestations of labyrinthine vertigo are the reactive movements caused by vertigo, the totality of which is summarized as objective vertigo. The reflex movements are sequelæ of reflex muscle innervation, and their principal purpose is to restore the equilibrium which has seemingly been disturbed by the rotatory vertigo.

If the reactive movements are sufficiently pronounced, they will culminate in objective vertigo. Further reactive manifestations, which, however, occur only in very severe paroxysms of vertigo, are muscular spasms and vomiting.

In *objective vertigo* there is always an acute reflex change of the muscle tonus, caused by the labyrinth. The tonus of the labyrinth is probably produced by the labyrintho-cerebral tracts, and its absence is followed by impairment of the coarse forces and faulty precision of movements.

Labyrinthine vertigo is always associated with nystagmus. If labyrinthine nystagmus persists for a long time, especially if its intensity remains unchanged or is but slowly reduced, vertigo is arrested after a relatively short time, while the nystagmus will persist for many weeks or months longer. Both intensity and movements of labyrinthine nystagmus vary in individual cases; nor can any general statement be made as to the patient's becoming accustomed to nystagmus.

The further consequences of objective vertigo are disturbances in equilibrium and gait, which can seldom be tested clinically, for the reason that the very presence of vertigo prevents patients from walking or even standing. However, we may summarize the characteristic equilibrial disturbances of the labyrinth as those which occur independently of vertigo and are persistent in character.

Labyrinthine vertigo is differentiated from the optical form in that the latter is immediately arrested by closure of the lids, in many cases by merely relieving or abandoning fixation, whereas labyrinthine vertigo is but slightly reduced by closure of the lids, if at all. Although neurasthenic or neurotic vertigo may sometimes simulate the labyrinthine form, there are no insurmountable difficulties in their differentiation. Labyrinthine vertigo is greatly dependent upon the position of the head, and occurs in affections of the labyrinth on arising in the morning and changing the recumbent position into the erect. The movements of the head in washing the face are likewise apt to produce this form of vertigo. The neurotic form, on the other hand, is independent of the position of the head, or, at any rate, is not so mechanically dependent upon it as labyrinthine vertigo. The neurotic form, combined with other nervous symptoms (oppression, etc.), preferably occurs in the evening after physical and mental fatigue, or perhaps in the street during the day.

Vertigo is sometimes complained of in cardiac affections or anæmia. These manifestations, however, amount, as a rule, to fainting fits rather than vertigo. In most cases patients describe the ground as giving way or having undulatory movements, or they complain of blackness before their eyes. The attack is usually produced by muscular work or physical exertion. Every paroxysm of cardiac vertigo is associated at least with transitory impairment or else complete loss of consciousness, while the sensorium remains undisturbed in the labyrinthine form.

Cerebellar vertigo is always associated with cerebellar ataxia and homolateral disturbances of coördination. It occurs in the shape of violent paroxysms, but any characteristic feeling of rotation either of the patient's own body or his surroundings has not been observed in this form.

Tabetic vertigo requires careful judgment, as in many of these

cases there is not only an affection of the segmental nerves, but also of the eighth nerve and labyrinth. There are degenerative changes of the labyrinth and auditory nerves in many cases of tabes, and it is therefore not surprising if these patients combine attacks of true labyrinthine vertigo and equilibrial disturbances with the peculiarities of the tabetic gait. In other tabetic cases, however, patients have vertigo without impairment of the labyrinth. Close questioning, however, shows that in these cases the expression "vertigo" is confused with "equilibrial disturbance," and careful examination (Romberg-Erben's test) will show that in so-called tabetic vertigo there is only a disturbance of the equilibrium, but no sensory illusion as to the topography of the body or its surroundings, provided the labyrinth is normal.

Vertigo may also be caused by the lower sensory organs (smell, taste, feeling). These are manifestations of neurotic origin or slight fainting fits of short duration.

A. EXAMINATION OF THE SEMICIRCULAR CANALS

The object of all functional tests is to establish the reflex excitability of the semicircular canals. The reflex symptoms of an irritation of that apparatus consist in nystagmus, vertigo, disturbed equilibrium, muscular spasms, and vomiting, and among these nystagmus has proved the most valuable for clinical examination, vertigo being only of inferior importance. The point, therefore, is to observe and interpret labyrinthine nystagmus produced by the irritation

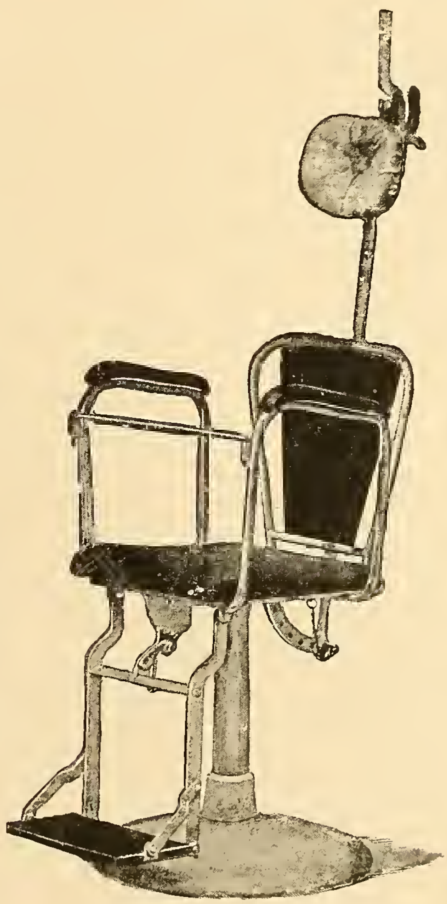
1. **Examination on the Rotatory Chair** (Fig. 49).—Formerly reflex vertigo was the object of the test; later it was found necessary to examine during rotation in order to find out the function of the semicircular canals,—*i.e.*, the patient had to rotate with the examiner, who had to make observations in spite of his own vertigo. At the present time we are able to use reflexes for diagnostic purposes which occur after rotation has been arrested, thanks to an exact study of after-vertigo and after-nystagmus. The rule is to rotate the patient on the chair about 10 times with increasing speed (positive acceleration), after which the motion is suddenly arrested. This will produce nystagmus to the left after rotation to the right, and nystagmus to the right after rotation to the left. There will be rotating nystagmus if the head is inclined toward rotation, or horizontal nystagmus if it is kept erect. In this experiment the normal duration of after-nystagmus rarely exceeds 15–40 seconds. Nystagmus to the left is nearly always referable to the irritated condition of the left labyrinth, and nystagmus to the right is referable to the right labyrinth. From this it follows that the rotation should be to the right for testing the reflex excitability of the left ear, and *vice versa*.

The degree of excitability is indicated by the duration of the after-

nystagmus. Normal excitability exists if the after-nystagmus lasts for 15–40 seconds in cases presenting no pathological symptoms of the static labyrinth. Shorter or longer duration of the nystagmus is individual and not pathological, provided there is no affection of the labyrinth and an examination of the opposite labyrinth leads to the same result.

In the presence of clinical symptoms of the labyrinth we speak of pathologically diminished excitability if the nystagmus lasts less than 15 seconds after 10 rotations, while the excitability is called prolonged (increased) if the nystagmus lasts for more than 40 seconds or if it lasts at all longer than the nystagmus of the opposite labyrinth after irritation.

Fig. 49.



Rotating chair.

Nystagmus produced by rotation is always examined in that direction of vision in which spontaneous nystagmus, if present, would not be visible, in order to prevent interference with the latter. This is best accomplished with straightforward vision. If there is intense spontaneous nystagmus, the direction of vision in which the spontaneous nystagmus cannot be observed is established before the examination. (Bárány's head-holder.)

Should there be no nystagmus after 10 rotations, the reflex excitability of the semicircular canals is regarded as extinct (excitability negative). Slight irritation of the opposite labyrinth, however, may in these cases cause slight nystagmus lasting for 6–8 seconds.

The great clinical importance of examination on the rotating chair is its simplicity and the utility of establishing functional irritations.

2. **In the caloric test** functional irritation is replaced by thermal irritation. Irrigation of the auditory meatus or middle ear with water below body temperature (70–85° F.) will cause rotatory nystagmus toward the opposite side, irrigation with water above body temperature (104° F.) toward the irrigated side. The time for the reaction to set in depends upon the condition of the tympanic membrane. If intact,

irrigation will have to be continued for some time; if destroyed, the caloric irritation will act immediately upon the lateral wall of the labyrinth and only short irrigation will be required. The reaction is explained by the fact that thermal irritation causes an endolymphatic current and an associated motion (oblique position) of the cupola, as it does on the rotating chair, and the direction of the current after elevation of temperature is opposite to that after cooling. The caloric test is valuable because it enables the examiner to test the semicircular canals separately on each side.

B. METHODS OF EXAMINATION OF THE SEMICIRCULAR AND VESTIBULAR APPARATUS

1. **The Galvanic Test.**—The semicircular and vestibular apparatus are tested by the galvanic current, which apparently excites all the rami of the nervus vestibularis and all the nerve-end places of the static labyrinth, so that in the present state of our knowledge we are unable to differentiate between the semicircular and vestibular apparatus by the galvanic test.

The test is made as follows: The cathode is placed against the tragus immediately in front of the external meatus or against the posterior margin of insertion of the concha. The anode is placed either in the nape of the neck, against the chest, or in the patient's hand. As the circuit is closed at an average strength of 4 ma., there will be a rotatory nystagmus toward the cathode; if the circuit is opened, there will be a short-beating nystagmus toward the opposite side. If the anode is at the ear, nystagmus toward the opposite side will occur on closing the circuit, and nystagmus toward the same side when the circuit is opened. The nystagmus should be established with straightforward vision, but it is easier if the patient is placed in the direction of the nystagmus and ordered to look away laterally. A current applied at greater strength than 4 ma. in a normal individual will cause objective vertigo, as evidenced by movements of the head and body and a tendency to fall. These movements occur in the opposite direction to that of the nystagmus, which means toward the anode. In cases of pathologically increased excitability of the semicircular canals the positive galvanic reaction may occur at 1 or 2 ma.

As a rule, the reaction will occur more rapidly and distinctly with the cathode at the ear than with the anode, the cathode-nystagmus being more distinct than the anode-nystagmus. In pathologically reduced or destroyed excitability of the labyrinth, the positive galvanic reaction will only set in at a current strength of from 6–13 ma., always provided the vestibular nerve is still capable of reacting upon the galvanic irritation ("trunk reaction" of Ewald). Should this no longer

be the case, the vestibular galvanic reaction will be negative, and this would indicate that not only the peripheral end-apparatus, or labyrinth, has lost its function, but the nerve also. The normal points of attack of the galvanic current are the sensory epithelium and the nerve. With intact condition of these two points, there will be normal reaction at an average strength of 4 ma.; with pathologically increased excitability of the labyrinth, the positive reaction will occur at less than 4 ma.; and with a pathologically reduced or destroyed excitability of the labyrinth, the positive galvanic vestibular reaction will occur at a current strength of 7–15 ma., as long as the vestibular nerve is still capable of conduction. With the vestibular apparatus and nerve destroyed, the galvanic reaction will be negative, and there will be no nystagmus even at the highest applicable current of 15–30 ma.

The galvanic test enables us to separate the condition of the peripheral apparatus from that of the nerve for diagnostic purposes, and this constitutes its particular value.

2. Examination of the Mechanical Reflex Excitability (Nystagmus from Compression and Aspiration, Kuemmel's Symptom of Pressure Variation, Fistular Symptom).—This test requires the use of a laterally perforated bulb, as in Gellé's test, or a small double bulb. The external auditory meatus is closed air-tight with the moistened bulb, when neither compression (elevation of pressure) nor aspiration (reduction of pressure) will produce a vestibular reaction, nystagmus or vertigo, under normal conditions. If, on the other hand, there is a fistular perforation of the osseous capsule of the labyrinth at a place accessible from the middle-ear spaces, both elevation and reduction of pressure will cause nystagmus. Nystagmus produced by compression is in the opposite direction to that produced by aspiration.

In typical cases the nystagmus thus produced consists of but few extensive twitchings, accompanied by considerable vertigo. In some cases pressing the tragus into the auditory meatus will sufficiently elevate the air pressure to cause the distinct fistular symptom. The mobility of the stapes is considerably increased by the destruction of the incus-stapes articulation, and this may suffice to cause nystagmus by compression and aspiration without there being a fistula in the labyrinth. In cases of congenital or prematurely acquired anomalies of the internal ear (syphilis of the labyrinth) nystagmus may likewise be produced by compression or aspiration.

C. METHODS OF TESTING THE VESTIBULAR APPARATUS

1. Testing the Equilibrium of the Body and the Disturbance of Vestibular Equilibrium.—The stability of the body is first tested by Romberg's method. The patient is then required to stand on one leg,

to walk forward and backward, and, if the patient is young, agile, and gymnastically trained, to hop forward and backward. Normal persons can perform these movements by the aid of the labyrinth, the eye, and the deep and superficial sensitiveness, these being the three components that go to make up the stability of the body. In order to accomplish these movements, however, two of these three components are sufficient, as in normal individuals standing, walking, or hopping is not impaired by closing the eyes. Even in pathological changes of the vestibular apparatus there may still be apparently good equilibrium, but if such an individual closes his eyes, there will be only one stability component —deep and superficial sensitiveness—to draw upon, and the defective equilibrium will be at once apparent. In cases of affected labyrinth, therefore, it is possible to establish the want of equilibrium by following this order of arrangement. This impaired equilibrium, however, has nothing to do with the temporary, secondary disturbances of stability caused by vertigo of the labyrinth.

2. **The Goniometer Test.**—The object of this test is to examine the stability of the body on an inclined plane. The patient stands with bare feet on the goniometer board, which is strewn with sand to afford a safe foothold. The patient assumes the Romberg position, and can thus, if normal, bear an incline up to 30° in any erect position, whether the board is raised in front, behind, at the right or left side (Kuemmel). If the equilibrium is moderately impaired without accompanying vertigo, there may be a decrease of stability with open eyes, but an angle of 30° can still be borne. Shutting the eyes, however, will at once disclose the defect. In some cases an incline of no more than a few degrees will be borne with eyes closed. The limit is reached when the patient can no longer keep his balance, seems liable to fall, or holds on with his hands to keep erect. For clinical purposes, therefore, the result of the test with open and closed eyes has to be taken into consideration. Nervous and frightened individuals may sometimes not be able to tolerate the maximum incline, but in these there will be no difference between the results with open and closed eyes; besides, the tolerance will often improve when the test is repeated.

3. **Test for Counter-rolling of the Eyes.**—In normal individuals counter-rolling of the eyes will occur when the head is laterally inclined. The vertical meridian of the bulbi remains unchanged with only moderate lateral inclination, while with exaggerated lateral inclination the simultaneous movement of the meridian is considerably diminished from the effect of counter-rolling. At an inclination of 45° counter-rolling amounts to 12–15° (Bárány), and slightly increases at a greater inclination. When the labyrinth is destroyed, counter-rolling is considerably reduced, to from 3° to 5°, or entirely suppressed.

Vestibular Disturbance of Gait

In destruction of both labyrinths there is permanent impairment of equilibrium, which causes the patient to adopt a broad manner of walking, which corresponds to the permanent positive Romberg test in cases of bilateral destruction of the labyrinth[1]; the object in walking with the lower extremities extended is to counteract the reduced stability following upon the destruction of the labyrinths. Deaf-mutes affect a dragging, noisy gait, as it assists their want of stability, and besides they are unable to hear the unpleasant noise their method of walking causes.

In patients with diseased labyrinths the disturbances of gait become more pronounced in the stage of vertigo. In violent attacks of vertigo patients are unable to walk or to stand, or sometimes to assume a sitting posture in bed. In the latter case they select a position which causes the least degree of vertigo. This is always the one in the direction of the least nystagmus, and they exhibit great anxiety to retain it. As long as these patients are able to walk about, any attack of vertigo will cause them to deviate from a straight course, especially when walking with closed eyes. At first they will always deviate in the direction of the nystagmus when their eyes are closed. In the presence of pronounced vertigo they will also walk in that direction with their eyes open. This tendency, however, may change after a time. They will half-instinctively correct the deviation after they have once become aware of it, and this may eventually enable them to walk in a perfectly straight line. Frequently, however, there is overcorrection which will cause them to deviate in the opposite direction, while in undercorrection there may still be slight deviation toward the direction of the nystagmus. In cases of pronounced vertigo the correction will often change from one side to the other, leading to a more or less pronounced zig-zag walk.

Labyrinthine and cerebellar disturbances of gait may be differentiated by the flank-walk test. Grasping the patient gently by the arm, we cause him to side-step with closed eyes. This can be accomplished toward both sides in patients with diseased labyrinths, while in cerebellar affections the flank walk is considerably disturbed toward the side of the affection. Thus, patients with affections of the right cerebellum will only with difficulty carry out the side-step to the right; their step is uncertain, there is danger of their falling down, being unable to make an initial step with the right leg.

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[1] The tracks made by the feet are recorded by letting the patient walk barefooted over a sheet of paper 6–8 yards long and 1 yard wide after the soles of the feet have been moistened with liquid paraffin. The outline of the fresh traces may be marked with a colored pencil. They will be still more distinct if the soles have been smeared with a mixture of soot and liquid paraffin.

Survey of the Acoustic and Static Tests

I am in the habit of making a record of the tests for functional findings by availing myself of the chart reproduced in Fig. 50. It provides for the findings of the hearing acuity (*V*, *v*, *P*), of the tuning-fork tests (*W*, *S*, *R*, *Gé*), of the Galton-pipe test (*Ga*), of the watch test through the cranial bones (*h*), of the hearing-tube test (*T*), and of Stenger's test (*St*). The last two are used in testing for unilateral deafness. The results of the functional tests of the static labyrinth are also marked opposite the corresponding abbreviations.

This table is intended to record the findings of the tuning-fork tests.

Fig. 50.

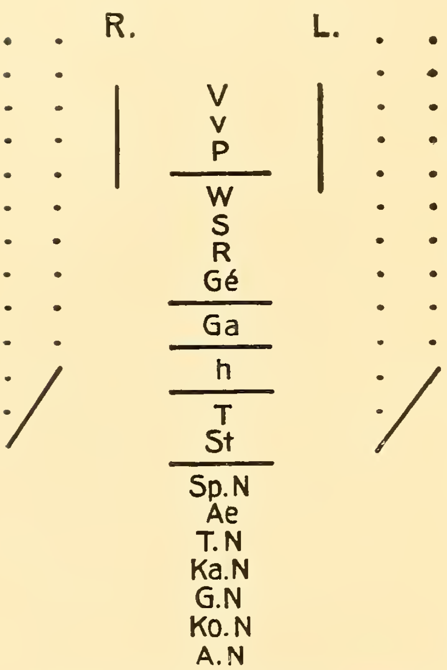


Fig. 51.

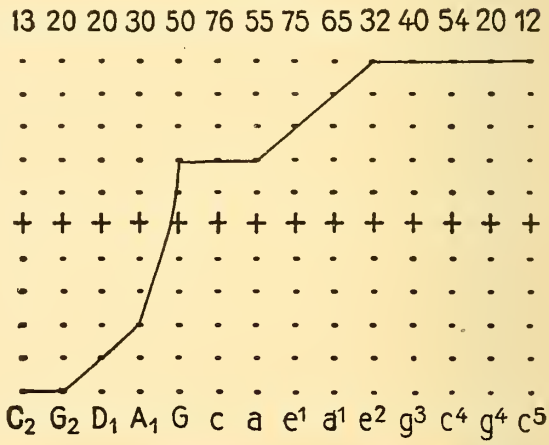


Fig. 50.—Table for dotting in the findings of the functional tests. *V*, conversation (Vox); *v*, whispering (vox); *P*, Politzer's acoumeter; *W*, Weber's test; *S*, Schwabach's test; *R*, Rinné's test; *Gé*, Gellé's test; *Ga*, Galton pipe; *h*, watch test (hora) through the cranial bones; *T*, hearing-tube (Tube); *St*, Stenger's test; *Sp. N*, spontaneous nystagmus; *Ae*, disturbance of equilibrium (Aequil); *T.N*, nystagmus after rotation (Torsion); *Ka.N*, caloric nystagmus; *G.N*, galvanic nystagmus; *Ko.N*, compression nystagmus; *A.N*, aspiration nystagmus. The findings for the right and left ear are dotted in under the letters R and L. The two vertical lines serve to indicate the hearing distance both before and after physical treatment (air-douche, catheter). The method of dotting in the results of Rinné's test will be understood from the remarks on pages 72, 73, and from Fig. 48.

Fig. 51.—Graphic representation of the quantitative tuning-fork tests based on Bezold's "acoustic relief."

The letters designate the various tuning-forks of the Bezold-Edelmann series. The figures indicate the normal duration of perception in seconds, by giving the forks a maximum blow (C_2 and e^2 weighted). Shortening of the duration of perception by one-half is indicated by +, while the dots represent shortening by 1-10th each.

The example selected represents the findings with a medium obstacle of conduction. C_2 and G_2 could not be heard, D_1 was heard for 2 seconds instead of the normal duration of 20 seconds, equal to a shortening by 9-10ths, etc. The sounds e^2–c^5 were heard for normal periods.

VII. LOCAL ANÆSTHESIA OF THE EAR

Anæsthesia of the Ear.—There is no need to emphasize the importance of anæsthetizing the external and middle ear, and it is useful to combine this proceeding with the induction of anæmia. Susceptibility to pain differs with the various parts of the ear, according to whether any particular part is normal, acutely inflamed, or recovering from some affection. Paracentesis can be almost painlessly carried out with a non-inflammatory tympanic membrane, for instance in suppurative otitis media, without requiring any particular preparation, and the same holds good for the resection of the posterior fold and other procedures, while paracentesis with an inflamed tympanic membrane is exceedingly painful without local anæsthesia. Susceptibility to pain in the membranous part of the external auditory duct does not differ from the rest of the skin, while the osseous part of the duct is even normally very sensitive to pressure or puncture. Fröschels has called attention to the possibility of subnormal susceptibility of the external auditory canal and the absence of the tickling reflex in otosclerosis. The normal and non-inflamed mucosa of the middle ear is but slightly sensitive to pain; exposed bone is completely insensitive.

Generally speaking, it is easier to induce anæsthesia when the epithelial layer of the mucous membranes has been destroyed; also in the presence of granulations, as compared to intact epithelial layers; in chronic as compared to acute cases of inflammation. In acute inflammation of the tympanic membrane, anæsthesia takes better effect after treatment of the affected parts with acetic alumina or Burrow's solution, but the reverse is the case after treatment with carbol-glycerine, the reason being that the first two remedies loosen and macerate the epidermal layer of the tympanic membrane.

Anæsthesia of the middle ear is easiest to induce in the meso- and epitympanum, more difficult in the hypotympanum, especially if the pathological changes have spread downward. Anæsthesia of the antrum and the tympanal ostium of the tube presents the greatest difficulties.

Local anæsthesia may be induced in three different ways: (1) instillation of fluid media, (2) insufflation of powders, and (3) injections.

For anæsthesia by instillation we use 5–20 per cent. solutions of cocainum hydrochloricum or its substitutes (novocaine, alypin). The solution is heated to 104°–108° F., and 5 drops of adrenalin solution (1 : 1000) are added to 1 c.c. shortly before use. When using the 20 per cent. solution, a cotton plug should be saturated and pushed forward to the part to be anæsthetized. Novocaine and alypin have the

advantage of being non-toxic. This method is particularly suitable in anæsthesia of the tympanic membrane if the epidermal layer of the latter is intact, also for short operations and paracentesis.

The anæsthetic effect of cocaine or its substitutes may be enhanced by electrolysis, with the anode in the external auditory canal. Insertion of a cotton plug saturated with adrenalin will not only insure ideal anæmia, but also deaden the pain, in some cases inducing complete anæsthesia. All the remedies mentioned are allowed to remain in the duct for 10–15 minutes. For the middle-ear spaces the instillation method is not suitable.

Insufflation of powders (novocaine, alypin, anæsthesin, cycloform) can be recommended for absorption of granulations. The traumatic pain following endotympanal operations may be easily overcome by insufflation of anæsthesin.

Satisfactory, complete, and long-lasting anæsthesia of the middle ear can only be accomplished by injection of 1 per cent. novocaine or alypin solutions, or Schleich's solution "I" (cocaini hydrochlorici 0.2, morphini hydrochlorici 0.02, acidi carbolici gtt. v, aquæ destill. ad. 100). The solution is heated to body temperature shortly before use, and 5 drops of adrenalin solution are added to 1 c.c. The most suitable syringe is Neumann's and the injection is made subperiosteally at the union of the cartilaginous and osseous parts of the duct. The upper wall of the duct is injected to anæsthetize the upper tympanic cavity, the postero-superior wall for the antrum, the basal wall for the hypotympanum, and the anterior wall for the tympanic end of the tube. The injection is made slowly and with little pressure, so as to prevent tearing the integument. A cotton plug saturated with the anæsthetizing fluid (preferably 20 per cent. novocaine or alypin) is inserted after the injection, and the patient allowed to rest for 10 to 15 minutes on the healthy ear.

In this way complete anæsthesia and anæmia of the middle ear are attained, which will last for about one-half hour. The Freiburg Ear Clinic recommends injections of a few drops of a 5–10 per cent. cocaine and adrenalin solution into the tympanic cavity to anæsthetize the tympanic membrane for paracentesis. Bernd recommends a thin, graded record syringe with a very fine platino-iridium needle. For removal of polypi, Frey recommends injection of Schleich's solution.

Many operators prefer to use ethyl chloride as a general anæsthetic, especially in children. Fortunately, the young are quickly anæsthetized, and as they rapidly recover from the effects, it is the best anæsthetic for short operations. A thin towel or a chloroform mask is held over the patient's face, and the spray striking the mask is quickly vaporized. The patient is in the analgesic stage before completely anæsthetized so that a myringotomy may be performed with deliberation and ease, not possible under local anæsthesia, especially in an over-wrought patient.

VIII. DISEASES OF THE CONCHA AND THE EXTERNAL AUDITORY MEATUS

1. CONGENITAL ANOMALIES OF DEVELOPMENT OF THE CONCHA AND THE EXTERNAL AUDITORY MEATUS

Slight deviations from the normal form and size of the concha and auditory meatus are very common. They include faulty or arrested development of the borders and end-parts of the auricle (tragus, helix, lobe), abnormal excrescences (Darwin's point, Macacus point), and the abnormal development of the concha itself (supernumerary crus anthelicis, flat or unusually small cymba conchæ, absence of antitragus, colo-

Fig. 52.

Coloboma of the concha with asymmetry of the skull and micrognathia (smallness of the jaws) in a twelve-year-old girl.

boma (Figs. 52 and 52*a*). In macrotia the concha is enlarged in all its parts, the cartilage being unusually broad (Fig. 54), the fossa navicularis unusually deep, and the posterior wall of the cartilaginous auditory meatus unusually long.

The consequence of these deformities is that in MACROTIA the concha is laterally deflected, and the anterior aspect is far more disturbing from a cosmetic point of view than the lateral aspect (Fig. 53). This condition occurs much oftener bilaterally than unilaterally, both conchæ being usually enlarged to the same extent and in the same form.

Treatment.—The patients' wish is to be freed from the disfigurement of pronounced macrotia, as it is often the cause of being teased in school, and sometimes interferes with their future career. The treatment consists in the surgical reduction of the concha and those parts which are most prominent in the anterior aspect, the latter being the most disfiguring. This refers particularly to the abnormally enlarged lobes, the lateral surface of which is displaced forward (Fig. 53). It is trimmed down until its anterior aspect is normal. The reduction of the concha itself is best accomplished by extirpation of falciform pieces of integumental cartilage and subsequent primary suture. Parts of the free border of the helix, if over-developed, can often be resected with excellent cosmetic results.

Fig. 52*a*.

Anterior aspect of case shown in Fig. 52.

In congenital microtia the concha is only rudimentary, owing to defective development of the auricular cartilage. In most cases of microtia there is also atresia of the external auditory canal. The entire picture is one of arrested development, primarily due to circumscribed adhesions of the amnion to the cranial epidermis in the auricular region, which occurs as early as the 4th to 8th embryonal week. At this period the free fold of the ear normally develops from fusion of the posterior and superior auricular prominences, while in cases of malformation the fold is either not developed

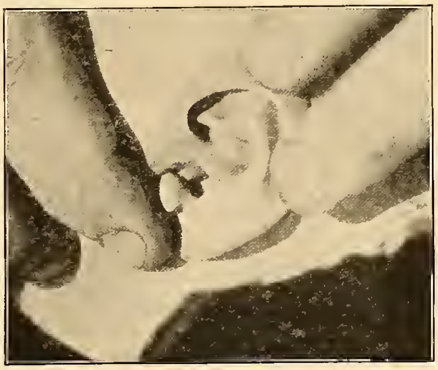
Fig. 53

Macrotia with disfiguring enlargement of the lobe. Boy fourteen years old. Cured by plastic operation.

at all or it is convoluted forward and downward instead of spreading backward and upward.

The extent of microtia is very variable, ranging from arrested development of certain parts to complete absence of the concha. In the latter case the concha is merely indicated by shapeless appendages of skin and fatty tissue.

Fig. 54.



Macrotia with accessory tragus. Boy three weeks old.

Congenital appendages (Figs. 55, 58, 59) in the auricular region and pre-auricular fistulæ (Fig. 56) are not very rare, particularly in the parotid region and auriculo-oral line, while appendages at the free parts of the ear itself are less common. Thus, falciform appendages of skin and cartilage are only exceptionally observed at the helix in the region of Darwin's point, the disfigurement being usually bilateral.

In making a differential diagnosis, it should be noted that skin tubercles in the auricular region in children may be mistaken for auricular appendages (Fig. 115).

Fig. 55.

Macrotia with multiple pre-auricular appendages. (Left side of Fig. 58.) Boy one year old.

Treatment.—Auroplastic operations are only applicable in cases where the cartilage has

Fig. 56.

Macrotia with micrognathia and multiple pre-auricular fistulæ in boy three months old.

developed to a sufficient extent, rendering the use of pediculated skin flaps possible from the direct vicinity of the ear, particularly from the planum mastoideum, for the enlargement of the ear fold, for-

mation of a helix, etc. Paraffin injections are in some cases fairly successful. Before injecting paraffin, a test injection is made with Schleich's solution I, to show the result to be expected, and, if satisfactory, small quantities of paraffin are injected at various sittings. Following Gersuny's instructions, I always use ung. paraffini, which is of ointment consistency at room temperature.

In supplementing the cosmetic procedure by surgical removal of the auricular appendages, great caution should be exercised, for the resulting cicatrix in individuals with a tendency to callous scar-formation may be more disfiguring than the original condition. Plastic operations should not be undertaken before the 6th to 8th year of age.

Protheses may be resorted to in considerable deformity of the concha. There are excellent soft rubber concha protheses on the market which are fastened with wax to the auricular rudiment. In the majority of cases, however, patients prefer hiding the defect by the hair.

2. CONGENITAL ATRESIA OF THE EXTERNAL AUDITORY CANAL

Congenital occlusion of the external auditory meatus is nearly always associated with congenital deformity of the external meatus, and is usually a part manifestation of a more or less important malformation of the external and medial ear.

Anatomy.—Congenital atresia develops as early as the second embryonal month, when the development of the tympanum in the mesoderm surrounding the capsule of the labyrinth normally sets in. If the rudimentary tympanum fails to develop, the immediate consequence is the close proximity of the submaxillary articulation to the pars mastoidea. The normal external meatus also fails to develop in these cases, and the skin at this place is either smooth or forms a short blind fossula from a few mm. to 1 cm. long. This deformity is usually associated with an irregularity of the concha, which may be either entirely missing or replaced by several small shapeless appendages. The cartilage is completely absent in some cases, while in others it is more or less rudimentary, serving as a support to the misshapen concha (Figs. 57, 58). Cases of congenital atresia with but slight deformity of the concha are rare (Fig. 59). Examination of the temporal bone will usually show a greatly enlarged pneumatic mastoid process in close proximity to the fossa articularis of the submaxillary bone.

Fig. 57.

Congenital atresia of the left external auditory duct, with rudimentary development of the concha.

There are in all cases characteristic changes of the middle ear, the lateral wall consisting of a compact osseous plate. The Eustachian tube is present in the majority of cases; in a smaller number, in which there is atresia of the tube, the musculature of the soft palate is imperfectly developed at the side of the deformity. The tympanic cavity is small, stenosed from osteophytes, becoming narrower toward the tube, and giving the impression of the osseous tube being continued much further into the tympanic cavity than normal. The osseous tube usually extends backward and outward to the corner of the cochlear window. The antrum is small. The middle wall of the tympanic cavity is in

Fig. 58.

Congenital atresia of the external auditory duct with microtia, the cartilage, however, being well developed. Auricular cone-shaped appendage in the auriculo-oral line; on the left side of this case (Fig. 55) there were macrotia and multiple auricular appendages. One-year-old boy.

most cases of normal shape, but there are often osseous defects of the facial canal, osseous deposits and connective-tissue ligaments which may cause stenosis or occlusion of the corners of the vestibular and cochlear windows. The auricular vessels and the tympanic membrane are demonstrable in most cases, but always exhibit grave changes, the tympanic membrane, in the shape of an irregularly demarcated, flabby membrane, being more or less accumbent, but not adherent, to the lateral osseous covering-plate of the middle ear. The auricular vessels are small and coarse. The head of the malleus or the long crus of the incus is often deformed, both crura of the stapes plate are fused into each other, and this, together with the stapes rudiment, often recalls a similarity with the columella of birds. Sometimes the head of the malleus is jointed to the body of the incus, and the medial auricular ossicles consist of the long crus of the incus, which at both its ends is united by connective tissue with the lateral and the inner ossicle, re-

spectively. The entire chain of ossicles is enveloped in a dense net of connective tissue. The muscles of the middle ear, though present, show considerable degeneration, while the chorda tympani is missing.

The contents of the middle ear more or less approach the normal in cases of slight deformity, while in higher degrees of malformation the rudimentary tympanic membrane may be completely absent, and, together with it, malleus and incus, nothing but the rudimentary stapes being demonstrable. The ligaments and connective tissue of the middle ear show in all cases stronger development than normal.

Fig. 59.

Congenital atresia of the external auditory duct with defective development of the helix ascendens (*a*) and a pre-auricular appendage (*b*). (Natural size.)

The internal ear is normally developed in most cases of congenital atresia of the auditory canal and arrested development of the middle-ear spaces, participating but seldom in the malformation. Where, however, the auricular deformity is associated with severe congenital malformation of the entire body,—notably of the head, brain, heart, arterial blood-vessels, nose, eyes, or in twin monstrosities, such as syncephaly, synotia, anencephaly,—there is, aside from atresia, congenital malformation of the labyrinth and auditory nerves. These cases, however, are of no clinical interest, the fœtus being either stillborn (usually prematurely) or dying a few hours after birth, owing to the grave general defects of development.

On the other hand, arrested development in congenital deafness is usually limited to the labyrinth, extensive or grave anomalies of the external or middle sections being but rarely observed. Extensive defects of development of all three auricular sections are a clinical rarity.

Symptoms.—Congenital atresia of the external auditory meatus is associated with a cosmetic imperfection and derangement of hearing caused by the obstacle to conduction.

Patients are generally not much concerned about the cosmetic imperfection, as they can cover any such defect by the hair, while disfiguring scars at any other place, as for instance at the cheek or neck, are far more unpleasant. The hearing distance is reduced in all cases, amounting to 2½–27 feet C., which is but rarely exceeded, as the cause is not only atresia of the external meatus, but also such congenital changes of the middle ear as have been described. Relatively good hearing distance points to a good condition and slight congenital malformation of the middle ear and to a permeable tube capable of physiological function.

Youthful patients generally experience no subjective complaints, while adults mostly complain of numbness in the head and a feeling of fulness in the ear.

Preservation of the middle ear in congenital atresia and defective concha may be the cause of suppurative otitis media, outward escape of the pus being impossible and escape through the Eustachian tube into the throat being impeded by the inflammatory swelling and constriction of the tube. This explains why the suppuration will spread to the mastoid process in a comparatively short time, perforating either outside through the periosteum or toward the dura into the cranial cavity, unless there is timely surgical aid.

The functional test in congenital deformity of the concha shows all the signs of a grave obstacle to conduction. Labyrinth symptoms are either entirely absent or there is a moderately reduced perception of high sounds with a lowered upper sound-limit, all of which points to changes in Corti's organ at the level of the promontory and cochlear window (vestibular part of the cochlea).

Treatment.—Congenital atresia of the auditory meatus can generally be noticed at the time of birth. Parents will ask whether and to what extent the misshapen ear may be expected to hear, and the satisfactory reply may be given in nearly all cases that there will be positive though reduced hearing ability.

The hearing distance may amount to 3–37 feet C. and 8–12 feet for the acoumeter, but may be materially improved by insufflation of air or catheterizing; many patients acquire improved hearing acuity through their perfectly normal or even prolonged bone-conduction. Several years ago I observed a boy of thirteen with bilateral congenital atresia whose hearing distance attained 7–10 feet C.; by pressing the forehead against a wooden board, an open door for instance, he was able to hear at a distance of 30–40 feet C.; indeed, merely touching him increased his hearing acuity. According to his statement, he could understand a person better by placing his hand on the shoulder or head of the person he was conversing with.

Cosmetic improvement seems impossible, since the submaxillary articulation immediately continues the mastoid process, and the middle-ear spaces are laterally closed by a compact bony layer. It is impossible, therefore, to construct an artificial auditory canal, extending to the tympanic membrane, which would correspond to normal conditions in regard to position, length, and course.

The hearing acuity may be surgically improved by antrotomy, provided the antrum can be kept open exteriorly by skin plastic. This operation should only be resorted to if there is a sufficiently large rudiment of the concha, bilateral atresia, and provided the preserved orifice

of the antrum is covered or surrounded by the rudiments of the concha. In unilateral atresia patients are usually satisfied with the functional use of the healthy ear. The plastic flaps are best taken from the skin of the mastoid. Improvement in these cases is quite perceptible and permanent. I have carried out this operation in three cases of congenital atresia in which antrotomy was indicated by an ulcerative process of the mastoid. Considerable and permanent improvement resulted in all three cases.

3. ACQUIRED DEFORMITY OF THE CONCHA AND EXTERNAL AUDITORY MEATUS; ACQUIRED POSITION ANOMALIES OF THE CONCHA

All inflammatory affections of the concha, primarily involving the cartilage and perichondrium or spreading from the integument to the perichondrium, may heal, but leave a more or less extensive deformity of the concha behind. Similarly, all skin defects of the concha lead to a deformity of the latter if they terminate in a keloid. Acquired pathological constriction (stenosis, stricture) of the external duct and acquired occlusion of the meatus (atresia) can nearly always be traced etiologically to deep circular ulcerations of the external duct which have persisted for a long time. Organic strictures of the auditory duct which develop in the course of chronic middle-ear suppurations also belong to this class.

Inflammation of the concha and suppurative perichondritis are regarded as the principal causes of acquired deformity of the concha, while tuberculous and serous perichondritis and hæmatoma auriculare are less often responsible. Where the cartilage is not involved, the deformity consists only in a scar which flattens the structure, the concavities of the concha disappearing by being more or less filled up with thick cicatricial tissue. Or there may be one or more keloid tumors protruding above the level of the concha, which are very disfiguring. Deformities occurring after corrosions of the concha can be primarily traced to perichondritis produced by trauma or to the keloid which developed in the healing process. Piercing the lobe for ear-rings may sometimes give rise to disfiguring keloids in little girls.

Inflammatory involvement of the cartilage may lead to circumscribed or diffuse necrosis, and auricular cartilage once destroyed will not be regenerated. The skin duplicature which is kept distended by the cartilage becomes flabby as the ear shrinks. This may lead to considerable deformity, in rare cases even to complete destruction of the concha, leaving merely a shapeless protuberance of thick, callous, cicatricial tissue invested with the epidermis of the concha (Fig. 60).

The treatment of acquired deformity of the concha is only cosmetically important. Injections of thiosinamin or fibrolysin may give

some degree of satisfaction and prevent subsequent calcification or ossification. Keloids which are well demarcated, protruding or crested, may be removed, the trauma being covered by Thiersch's flaps. In this way it is usually possible completely and permanently to remove these disfiguring protuberances.

Paraffin injections are in many cases of acquired corrugation of the concha cosmetically fairly successful. The first step in this process is to make a tentative injection with Schleich's solution, in order to observe the cosmetic effect to be expected, and, if the contour is improved

Fig. 60.

Shrinking of the concha after suppurative perichondritis.

and the free parts of the shrunken ear are enlarged backward and upward, the case is a suitable one for paraffin injection. This should be done very carefully at several sittings, using the smallest quantities (not exceeding $\frac{1}{4}$–$\frac{1}{2}$ c.c.) at one injection.

This treatment is suitable in all cases of perichondritic corrugation of the concha in which the otherwise deformed cartilage is not bent laterally; otherwise paraffin injections will not be able to distend the parts backward and upward. In these unsuitable cases paraffin would simply distend the skin laterally so that the enlarged remnant of the concha would stand off from the head, or the paraffin deposit would form a lateral tumor-like protrusion. In lateral bending of the cartilage, paraffin injections should, therefore, be entirely refrained from.

In many cases of deformity of the concha, corrosion of the auricular region causes a flat fusion of the middle plane of the concha with the mastoid and temporal regions. This can be remedied by circumcising the concha and remobilizing the free part with the aid of pedunculated flaps. The skin of the mastoid region is preferably used for this purpose, or, should this be cicatrized, the skin of the upper neck or nape. The operation is best done at several sittings. Using, as I am in the habit of doing, 3 right-angled flaps, the upper part of the concha is mobilized at the first sitting, the lower one at the second (formation of the freely movable lobe), and the medial part at the third. Adhesion of the operated parts is prevented by insertion of a double flap of epidermis.

4. ACQUIRED ATRESIA OF THE EXTERNAL AUDITORY MEATUS

Acquired atresia is either membranous or solid. The solid form may consist of connective tissue or bone, or both.

Acquired atresia of the external meatus results from (1) trauma (cuts splitting the external meatus and severing the concha, fracture of the external duct, corrosions); (2) chronic ulceration of the external meatus; (3) croupous inflammation of the external meatus; (4) bone formation in the middle ear in the course or at the end of chronic suppuration of the middle ear; (5) tumors.

Cuts completely splitting the external duct and severing the concha are rare. They are almost exclusively sabre cuts hitting the parotid region in a vertical direction.

The weight of the concha causes the lobe to gravitate downward even though the wound has been sutured in time. In the healing process the severed outer end of the external duct will be located below the level of the medial one, with the result that the medial part will be occluded by the cicatricial mass externally and the lateral part internally.

Atresia in these cases can only be prevented by making a gutta-percha cast of the healthy meatus and holding the severed parts in proper position with its aid.

Fractures of the auditory meatus caused by great violence (kicks from a horse or other blunt blows) may likewise cause atresia while healing, by dislocation of the fractured parts. In slight dislocations the meatus may assume quite a peculiar shape. I observed a case in which the kick from a horse led to fracture of the parietal and temporal bones and external meatus. The otoscopic picture of the latter represented the figure 8, the upper ring of the figure being situated anterosuperiorly and the other postero-inferiorly; they communicated with each other. This shape was caused by the fractured parts of the osseous auditory duct having become dislocated and healed with callous exostotic eminences (Fig. 61).

Atresia due to corrosions is caused by the corrosive agent penetrating into the external meatus leading to complete or at least annular destruction of the integument. They are usually the result of assaults in which large quantities of the corrosive fluid (solution of potash, sulphuric, carbolic, or nitric acid) are thrown against the ear at a short distance, so that a portion of it will find its way into the meatus. Self-inflicted injuries of this kind are rare.

Local treatment, though immediately instituted, will usually be unable to prevent atresia, and the ulceration is favored by the middle-ear suppuration set up by the same trauma.

In atresia caused by croupous inflammation or ulceration of the auditory duct, or by sequestration of the osseous duct, there is nearly always chronic, fetid, neglected suppuration of the middle ear, the stagnation of the fetid pus in the external meatus leading to maceration of the investing epithelium and ulcer formation, especially in the region of the osseous portion of the duct. The more frequent course, however, is for atresia to develop with periodical arrest of the ulceration of the auditory duct, the middle-ear suppuration continuing and occluding the external meatus for several weeks or months. As soon as the pressure of the secretion in the middle ear is strong enough or a cholesteatoma presses against the occlusion, the latter will be perforated, allowing pus and cholesteatomatous matter to escape. This fistula may become permanent or be closed from time to time. There is, of course, the danger of a fresh perforation occurring toward the interior of the cranium, involving the endocranium. This process explains cases of atresia in which, after a prolonged period of well being and entire cessation of the external ulceration, there occur sudden symptoms of intracranial complications.

Fig. 61.

Conical exostosis of the external auditory duct following fracture of the duct. Tympanic membrane normal.

There are cases of radical operation in which the trauma heals with connective-tissue atresia of the middle ear, which may extend to the level of the former external meatus.

Formation of osteophytes in the course of chronic suppuration of the middle ear may lead to osseous occlusion of the middle ear and osseous atresia of the external meatus if the integument of the osseous part of the duct has been previously destroyed.

Tumors of the bones and cartilage, as well as sarcoma, may extend to the auditory canal, leading to its complete obliteration. In the first form of tumor we have to deal with very rare osteomata of the mastoid process, otherwise merely with tumors of the submaxillary articulation; in sarcoma, with tumors of the parotid (Fig. 113), of the middle ear, or of the superior maxilla.

Symptoms.—If atresia occurs at the level or in the vicinity of the external auditory meatus, the patient will feel the occlusion with his finger and is soon aware of the cosmetic injury arising from the absence of the orifice. In all other cases, the manifestations depend upon the condition of the middle ear prior to the occlusion. With a healthy middle ear, atresia causes a more or less important reduction of the hearing distance, sometimes a sensation of fulness in the ear. On the other hand, atresia occurring in the course of chronic suppuration of the middle ear may be without symptoms, as the hearing acuity is already injuried to such an extent that any further exacerbation remains unnoticed. But in cases where suppuration of the middle ear persists behind the occlusion, patients will complain of regional pains, tinnitus, headache, sensation of heaviness in the head, and evacuation of pus through the Eustachian tube into the throat; besides, the pressure of the secretion may cause irritative manifestations of the labyrinth, such as spontaneous nystagmus, rotatory vertigo, equilibrial disturbance and vomiting.

The diagnosis of atresia of the auditory meatus presents no difficulties, the otoscopic picture showing the fossular, epithelialized occlusion. Less experienced physicians may have some difficulty in distinguishing membranous atresia from a cicatrized tympanic membrane, and for these the following guiding points will be helpful. In atresia the manubrium can be neither seen nor palpated, and, besides, the occlusion is nearer the eye than the normal tympanic membrane. Furthermore, the entire normal tympanic membrane, with the exception of its anterosuperior border, is demarcated by a straight, sharp line against the integument of the auditory canal. The wall of the latter and the tympanic membrane form an angle, one side of which has the pink color of the skin, and the other side the pearl-gray color of the tympanic membrane. An angular transition of this kind of the integument of the auditory duct is never found in atresia. In connective-tissue atresia the integument of the auditory canal fuses gradually with the membrane of the occlusion without forming a distinct angle, and the peripheral parts of the membrane generally admit of a distinc recognition of the color of the skin. Dubious cases can be decided by investigation with the probe and Siegle's speculum.

Treatment.—The only method of completely and permanently removing acquired atresia of the auditory meatus consists in plastic operations which correspond to the various types used in the radical operation. Thus, the auditory duct will be mobilized from the retroauricular skin flap, severing the blind end of the duct with the scalpel. This creates the type of entirely normal conditions, the only difference being that the duct will be much shorter than normal if the atresia is

close to the orifice. The simplest and best method is Koerner's or Sibenmann's plastic operation with tongue-shaped flaps. The skin flaps are thinned out as much as possible and fixed by catgut sutures. In simultaneous suppuration of the middle ear, the radical operation is, of course, combined with that for atresia. When the middle ear is healed, subsequent formation of cicatrical strictures of the external meatus should be prevented by resecting the corticalis up to a few mm. in the posterior osseous wall, the traumatic surface of the bone offering favorable conditions for rapid junction of the plastic flaps.

As a rule, the plastic cuts are made up to the orifice of the auditory duct, except when atresia is a sequel to traumatic splitting of the external meatus and gravitation of the lateral part of the duct; in that case the incisions should be made backward and upward into the cymba conchæ. It is impossible in these cases to return the concha from its abnormally deep adhesions to the normal position, and we must be content with directing the new auditory duct to the correct upward position, which is effected by enlarging the normal auditory meatus backward and upward to an extent which will enable us at least to survey the tympanic membrane from the upper part of the auditory canal in the otoscopic picture.

All other surgical methods recommended for atresia either fail from the beginning or will not prevent recurrence of the condition. Even in cases of membranous atresia the mere removal of the occluding membrane will not be permanently successful, as the subsequent cicatricial tissue invariably shows a tendency to recrudescence, so that re-formation of the atresia would be unavoidable. Furthermore, extirpation of fibrous atresia is not without danger, as injury to adjacent parts is extremely liable to occur, paving the way to traumatic meningitis or ulceration of the labyrinth (luxation of the stapes!).

5. TRAUMATIC INJURIES OF THE CONCHA AND THE EXTERNAL AUDITORY MEATUS, INCLUDING CUTS AND BITES

Contusions of the concha lead to skin hemorrhages, sometimes to extensive effusions of blood, at the lateral surface between the cartilage and perichondrium (hæmatoma auriculare). Generally speaking, hæmatoma is rare in children and is nearly always due to trauma. Hæmatomata are particularly liable to occur from violent blows, rarely from pulling or pressing.

The symptomatic picture of congelation of the concha corresponds to an acute dermatitis spreading to the perichondrium and is often associated with considerable swelling. Continued exposure to cold may lead to more or less extensive necrosis of the auricular cartilage.

Burns and corrosions of the concha present different clinical find-

ings according to the degree of the injury and the time of exposure. The slightest degree consists in hyperæmia and œdema of the skin of the concha, perichondrium and cartilage remaining intact. In serious cases the integument is destroyed, usually leading to acute perichondritis within a few days, with the ulcerative inflammation unavoidably spreading to the cartilage.

Burning or corrosion of the ear and vicinity lead to extensive superficial ulceration, the centre of which is the denuded, inflamed concha. The elasticity of the cartilage has been destroyed; the concha recedes almost completely against the cranium after the auditory duct has become ulcerative, which is usually the case.

In anæmic children suffering from a serious disease, pressure necrosis of the cartilage may develop (Fig. 62). This is almost without exception a sign of negligence, and occurs from allowing the patients to lie too long on one side, the head exercising continued pressure on the kinked and compressed concha. Ulceration can be avoided by cleanliness (bathing the ear with benzine and applying borvaseline to the inflamed places). Care should be taken that the concha is always flat, that the head rests on a soft pillow, and that the children should not remain longer than an hour in the same position.

FIG. 62.

Pressure necrosis of the auricular cartilage in a child three years old, with advanced pulmonary and glandular tuberculosis.

Symptoms.—The local pain is but rarely extreme and subsides rapidly even after serious injury. The temperature is normal or slightly elevated. According to the stage and degree of the injury, the concha is more or less swollen, the skin hyperæmic, raised by vesicles, or ulcerating. When extensive ulceration has already taken place, there will be all the signs of ulcerative perichondritis with abscess or ulcer formation.

The course of traumatic injury to the concha is entirely dependent upon whether the perichondrium and auricular cartilage have been involved. If not, the cosmetic result will be completely satisfactory. Involvement of the perichondrium, however, will probably lead to thickening of the concha. Partial destruction of the cartilage will nearly always cause shrinking, since the destroyed parts will not be renovated by cartilage, but by cicatricial tissue, the gradual shrinking of which cannot be prevented. In corrosion of the concha and the lateral cranial skin, superficial cicatricial adhesions between concha and cranium will develop, unless early cutaneous transplantation is resorted to.

Injury to the concha from cutting instruments, especially sabre

cuts, is very rare in childhood. Bites from dogs, entirely perforating the concha, including both the epidermal layer and cartilage, are more frequent.

The prognosis of cuts is cosmetically favorable, unless the injury is complicated by an ulcerative infection. Owing to the abundant anastomosis of blood-vessels and the excellent blood supply of the entire concha, the severed parts will grow together, even if only a narrow bridge is left to connect them and the concha is completely cut through. I have seen several such cases with excellent cosmetic results.

The treatment of tears and cuts consists in carefully cleansing the wound with a bichloride solution and correctly approximating the severed parts with a few cutaneous sutures; these should not go beyond the skin, so that perichondrium and cartilage will not be touched. Even if the concha has been completely severed, there is still a chance of success if correct apposition and fixation have been attended to at an early stage.

6. SEROUS PERICHONDRITIS

In serous perichondritis there is a clear, yellow, serous or seromucous exudate between the lateral surface of the cartilage and the skin of the cartilage. The favorite seats of the inflammation are the fossa of the helix and the triangular fossa. Accumulations of the exudate in the cymba conchæ are rare, while in the basal and medial parts of the concha perichondritis has never been observed. The inflammation occurs without any known cause or after slight trauma. Constitutional anomalies, such as anæmia and affections of the blood and blood-vessels, and chronic infectious diseases, such as tuberculosis and syphilis, seem to favor the occurrence of serous perichondritis. In patients suffering from serious illness, the affection may occur from the head lying too long upon the kinked concha, although circumscribed necrosis would be a more frequent consequence.

In the majority of cases the affection is unilateral, although its bilateral occurrence is by no means rare. It occurs principally toward the end of winter and is more frequent in northern than in southern climes, so that apparently cold weather favors its occurrence. Bacteriological examination reveals either no micro-organisms at all or only degenerated ones.

Symptoms.—There is neither pain nor elevation of temperature, as a rule, but in the initial stage there may be an unpleasant sensation of tension in the ear. Moderate elevation of temperature in the initial stage has been observed in some exceptional cases.

The diagnosis is made from the characteristic onset and further course of the affection. The only question to decide is whether there

is an accumulation of serous, sanguineous, or purulent exudate in the region of the swelling. This is determined by examining the diaphanous properties of the concha by means of a reversed monaural stethoscope. The concha is pushed away from the head and held against a light; in serous perichondritis it appears light red, in hæmatoma (accumulated blood) dark red, and in accumulation of pus the field of vision will be black.

The skin of the concha remains completely unchanged, even with extensive and long-persisting exudation: a symptom which allows of differentiation between serous and purulent or phlegmonous perichondritis.

Treatment.—The best cosmetic result is attained by conservative treatment, consisting in light massage with an iodine ointment or with Credé's silver ointment. Compresses saturated with absolute alcohol are applied overnight, the auditory meatus having first been closed by a firm cotton plug and a small strip of Billroth's gauze or gutta-percha. Under this treatment the exudate will be resorbed in the course of 4–8 weeks, leading in some cases to normal restoration of the concha and in others to a slight, cosmetically unimportant thickening of the cartilaginous skin in the region of the serous exudate.

During the last few years I have completely abandoned the surgical treatment of serous perichondritis. Aspiration and subsequent application of compresses have no effect, as within a short time, sometimes within a few hours, a new exudate will accumulate equal dimensions. Evacuation of the exudate with subsequent injection of irritating fluids (iodine solution, weak formalin solution, chromic acid solution) are to be deprecated, giving rise as they do to an apprehension of cartilaginous necrosis and transformation of the serous into purulent perichondritis with abscess formation. Incision and tamponade of the exuding cavity with iodoform gauze have been recommended; but the healing process under this treatment is very slow, and the patient is sometimes compelled to wear a bandage over the ear for several weeks, and the final outcome always shows perichondritic thickening with a callous cicatrix which is often of a very disfiguring character.

7. PHLEGMONOUS PERICHONDRITIS. ACUTE PURULENT PERICHONDRITIS

Acute purulent inflammation of the perichondrium and connective tissue of the concha occurs exclusively from infection with bacterial pyogenic factors. The bacillus pyocyaneus is found in the great majority of cases, although infection with the various kinds of streptococci, staphylococci and diplococci pneumoniæ is by no means rare. In exceptional cases, perichondritis may be caused by bacterium coli or proteus.

The conchial inflammation usually develops from a traumatic superficial injury to the concha. An inflammation may also develop from secondary infection after operation on the mastoid.

Symptoms.—The phlegmonous inflammation sets in with a diffuse swelling and reddening of the concha. The contour of the lateral surface becomes gradually obliterated. The concha continually increases in size and weight and may attain to twice its normal conditions. The skin becomes fissured, and finally desquamates in crusts or sclerotic, moist, epidermal rags. As a rule, pains only occur at the beginning of the affection, while medium elevation of temperature will persist for one or two weeks. Patients also complain of numbness of the head, heaviness of the entire auricular region, and lassitude.

Course.—In a small number of cases, phlegmonous inflammation of the concha will heal by spontaneous resorption of the inflammatory exudate without abscess formation; but in the majority of cases an abscess will form, in the course of the second week or later, at the lateral surface of the concha, in the area of the fossa helicis or cymba conchæ, and then purulent perichondritis will mark the end of the phlegmonous inflammation of the concha. Perichondritic abscesses of the medial surface of the concha are rare. The phlegmonous inflammation may also spread from the concha to the membranous part of the auditory duct, or a diffuse swelling may extend to the region of the upper neck and parotis, less often to the mastoid region.

The diagnosis is usually easy if the climax of the affection has been reached. In the initial stage it may be difficult to differentiate between it and erysipelas of the concha and herpes zoster. Besides, erysipelas is in many cases associated with phlegmonous inflammation of the concha.

In a painless and afebrile course of an apparent phlegmonous perichondritis there is always the suspicion of tuberculous perichondritis. In the latter affection there are one or more long fistulous canals which end superficially at the medial conchial surface near the lobe. In the absence of any such fistulæ the painlessness of the initial stage, normal temperature, and protracted course point to the tuberculous nature of the process.

Treatment.—In the initial stage of the inflammation the surface is covered with sterile borvaseline, and a moist compression applied with warm acetic alumina or 1 per cent. lysol. Above this a warm or cold compress (thermophore, ice-bag) is applied, according to the patient's liking. If there are signs of a spontaneous involution without abscess formation, 90 per cent. alcohol or salicyl alcohol compresses are applied, which have often a remarkably rapid favorable effect. An abscess should not be incised until it is distinctly demarcated and there is fluctuation in the entire surrounding region. Early incision is not to be

recommended because, in spite of the incision, other ulcerations may form and require renewed incisions. Care should be taken to make the incision from the medial plane of the concha, in order to prevent any disfigurement of the conchial contour by an unavoidable scar at the lateral surface. The incision is invariably made at the medial surface when the abscess is located there, but also in lateral abscesses which have reached considerable dimensions and led at least at one place to perforation of the necrotic auricular cartilage. The abscess can then be drained through the opening to the medial surface of the concha.

Should incision at the lateral conchial surface be necessary, it should be made to terminate anteriorly in a concave shape for cosmetic reasons, as the scars thereby occasioned will best fit in with the normal lines of the concha. Furthermore, it is advisable to incise at a concave place of the concha, so that the convex parts of the lateral plane may remain topographically unchanged after healing.

Result.—Any case of phlegmonous or purulent perichondritis may heal with a permanent deformity of the concha, which, if slight, will consist of a diffuse thickening of the concha resulting from the permanent thickening of the perichondrium, or there may be flattening of the lateral conchial surface or perpetual scars after incisions.

In necrosis of large parts of the perichondrium, shrinking of the concha after healing is unavoidable, and, in unfavorable cases with a grave course, all that may remain of the concha is a shapeless fibrous appendage covered with skin.

The patient, or those in charge of him, should always be informed at an early period, preferably at the time of making the diagnosis, that a permanent disfigurement may be expected after the inflammation has subsided; otherwise there may be reproaches later on, and possibly legal proceedings.

8. FOREIGN BODIES IN THE EXTERNAL AUDITORY DUCT

In most cases of foreign bodies in the ear, children have inserted them while playing, especially if they are in the habit of boring their fingers into the meatus because of chronic itching or eczema. It is comparatively seldom that one child stuffs foreign bodies into the ear of another. The objects vary considerably, and may consist of cherry-stones, coffee-beans, beans, peas, small pieces of wood, pearls, bits of lead-pencil, plant-seeds, little stones, pieces of garlic, cotton, etc. Living fly larvæ (musca or lucilia macellaria) and fungi (aspergillus, ascophora, and mucor mucedo) are sometimes found in the meatus. Small beetles, fleas, bugs, ear-worms, may find their way into the ears of children sleeping in the open or in unclean beds. Small winged insects, even small butterflies, may similarly intrude while buzzing about.

The first clinical question is whether the foreign body is one which will remain unchanged or undergo secondary changes. Beans, for instance, may gradually desiccate in the ear, but may also swell or even germinate in the ear, especially if water has simultaneously penetrated. Garlic particles may lead to painless hyperæmia (corrosion) of the tympanic membrane. A second point of clinical importance is the position of the article. An object introduced by the patient himself, and upon which no unsuccessful attempt at extraction has been made, will usually remain in the membranous part of the auditory duct. If the article is specifically heavy (small stones), it may wander into the osseous part, but will never transgress the isthmus. Only such articles will be found beyond the isthmus which have been inserted by others, or upon which unsuccessful attempts at extraction have been made.

Symptoms.—Articles which are not subject to secondary changes and are located in front of the isthmus may remain for a long time without giving rise to any symptoms. Children may forget the fact of their presence or omit to mention it; the articles will gradually be covered with cerumen, and may perhaps be found years afterward when a ceruminous embolus is removed or the ear is examined for some other purpose.

If the article occludes the auditory canal, it may form a considerable obstacle to sound-conduction and cause partial deafness, especially if there was an accumulation of cerumen which has been pushed forward against the tympanum. Impacted articles also occasion unpleasant tickling or pressure, and sometimes pain. The movement of living insects in the ear causes loud subjective noises. Articles impacted in the osseous part, in the isthmus, or in the deep parts of the external canal cause considerable pain; those which are subject to decomposition may lead to inflammation of the skin or of the entire canal. If the integument of the latter or the tympanic membrane has been injured, there will be bleeding from the ear, either immediately or in the course of a few days. Serious general manifestations and cerebral symptoms point to extensive injuries of the ear or of parts of the temporal bone adjacent to the cerebral cavity.

The most serious symptoms occur from unsuccessful attempts at extraction. Unsuitable instruments may cause extensive injuries, and every unsuccessful attempt will unfavorably change the position of the article, which is always pushed deeper into the ear, perhaps down into the middle ear. The use of an unclean instrument, such as a hairpin, toothpick, ear-spoon, etc., may lead to serious suppuration of the middle ear and consequent decomposition of the foreign body, particles of which may find their way into the middle-ear spaces. In favorable cases, however, the article will be expelled together with the pus or be entirely destroyed by the latter. Living larvæ of flies are found in the

external ear in many cases of neglected chronic suppurative otitis media, or fungi (otomycosis) in cases of eczema of the auditory canal or ceruminal emboli.

The diagnosis of the foreign body does not, as a rule, cause any difficulty, especially as children are usually brought to the physician with the express statement that a foreign body has entered the ear. If the auditory canal has otherwise remained unchanged, there will be no difficulty in establishing the nature of the article with the otoscope. This, however, is not sufficient, and the article should be palpated and diagnosed with the sound. This is the more valuable when neither the patient nor his parents are able to state definitely what the nature of the article is. A swollen meatus should be gradually widened by the careful insertion of small specula until it is possible to inspect the article.

Blood coagula preventing inspection should be removed with a cotton tip.

The prognosis is favorable in all cases in which the foreign body has been introduced by the patient himself, where there is no inflammatory reaction of the meatus, and where no unsuccessful attempts at extraction have been made. Statements in regard to the latter point are not always reliable, as those responsible for the attempts are apt to deny them. If the meatus is unchanged, and the child allows the examination to proceed without objection or fear, it may be taken for granted that the article has so far remained untouched. Should the child object, show fear, or complain of pain in the external meatus, should fresh or coagulated blood be found there, then there can be no doubt that attempts at extraction have been made, and responsibility for the possible consequences should be declined, as the tympanic membrane might have been ruptured in those attempts, and, if the physician subsequently injects water to remove the article, an inflammation of the middle ear may be the result.

In regard to hearing ability after the extraction, any statements made should be very guarded, since there is always a possibility of a previously existing affection of the ear. I am in the habit of confining myself to the statement, even in quite uncomplicated cases, that the child will be able to hear as well after the removal as before introduction of the article. It is a rare occurrence that chronic suppuration of the middle ear should have escaped the relatives, leading a child to stuff some article into his ear, but, if the physician has not given thought to this contingency, he may be held responsible for the suppuration when it reappears after the removal of the article.

Treatment.—Articles located in the membranous portion of the auditory canal can be removed without difficulty by the injection of water. The syringe should hold 150–200 c.c. and be provided with a

cannula from 2 to 3 mm. wide. The patient is directed to open his mouth, and the cannula is inserted into the auditory meatus so that the longitudinal axis of the syringe will point downward and inward. The concha is firmly pulled backward and upward, thereby stretching the auditory meatus. Impacted articles may likewise be removed with the syringe in many cases. If the article cannot be dislodged with the head in the ordinary position, supination with the head hanging down may facilitate successful removal. The water used should be sterilized and have a temperature of 100°–104° F.

Syringing is contraindicated if the article to be removed lies with a concave surface toward the meatus, if the article is decomposed or has undergone secondary changes (germinating beans, decomposed particles of garlic), or if the integument of the external meatus is inflamed and the auditory canal constricted.

In these and all such cases in which the article cannot be dislodged with the syringe, its instrumental removal should be considered. For this purpose firmly grasping, serrated, geniculate forceps or blunt and pointed hooks are used (Fig. 63). Large and small forceps should be held in readiness. Any such article as can be picked up from the hand without danger of slipping can be removed with the forceps, as for instance coffee-beans, pieces of bone or glass, intact pieces of garlic, paper, etc. If there has been a previous injection, the article should be carefully dried with cotton tips so as to prevent the forceps slipping. Round articles with smooth surfaces, such as little pebbles, cherry-stones, corn, etc., should not be touched with the forceps. These articles should be removed with the hook, should attempts with the syringe prove unavailing, blunt hooks being used for hard articles and pointed hooks for soft ones. The hook is inserted flat between the upper wall of the auditory canal and the foreign body, and then turned downward. Hard articles can then be brought out by careful gliding and rotating movements. For soft articles permitting the use of the hook, the instrument should be twisted by 90° while inserting it, the article being hooked while withdrawing it. It is then brought out with one traction of the hook.

Fig. 63.

Pointed hook for removal of foreign bodies. (Two-thirds natural size.)

In order to determine the position of the hook from outside, the instrument should be fitted with a four-edged handle appropriately marked, so that the position of the handle will always indicate the position of the hook inside the ear. Superficially located articles at the entrance to the canal which cause no occlusion may be easily removed with a small oval spoon made of metal or hard rubber.

Instrumental removal of a foreign body is a piece of art which requires considerable manual dexterity. The assistance of a trained nurse is requisite to ensure absolute stability of the patient's head, as otherwise an unexpected movement may cause injuries to canal and membrane with the pointed hook. In restless children or in the presence of an inflammatory process of the external auditory canal it is advisable to induce inhalation anæsthesia.

The removal of articles introduced with great force (projectiles, wood or glass splinters in explosions) and impacted in the osseous part of the canal may require operative interference. So do foreign bodies which have been firmly wedged for a long time deep in the osseous canal and beyond the isthmus. In these cases the osseous canal should be exposed by a retro-auricular cutaneous incision. Should this be unsuccessful, the posterosuperior wall of the auditory duct should be superficially removed with the chisel. This will widen the osseous canal and render the removal of the article possible. If the latter has penetrated to the mastoid process or antrum, opening of the mastoid or antrotomy will be necessary. Should the membranous portion of the duct be intact, it is readjusted after the article has been removed, and packed with xeroform gauze. In cases of extensive injury or ulceration, plastic surgery should be resorted to in order to prevent cicatricial stricture or atresia. In most cases it will be sufficient to make a horizontal incision along the posterior wall of the auditory canal. In considerable injury to the canal, one of the plastic methods used in the radical operation should be selected. Ulcerative parts are removed with the scissors. The plastic flaps are kept in position by catgut sutures and tampons.

Larvæ or flies can be extracted with ear-forceps after instilling a few drops of volatile oil into the meatus (turpentine, origanum). They sometimes attach themselves with their suckers to the middle-ear mucosa or to granulations. Small insects are removed with the syringe or, should they be otoscopically visible, with the forceps.

Iron articles can be removed with the powerful magnets used by oculists or with Hirschberg's electromagnet.

After removal of the foreign body, the exact condition of the tympanic membrane should be determined and recorded in writing. The auditory meatus is then closed by an antiseptic gauze strip. Should there be any apprehension of the ear being meddled with after operation (instillation of oil, etc.), an ear-bandage should be applied. It is also advisable at once to inform those in charge of the patient of the prognosis and to point out the possibility of middle-ear inflammation in the event of any injury to the tympanic membrane.

9. OTITIS EXTERNA ECZEMATOSA (ECZEMA OF THE CONCHA AND EXTERNAL AUDITORY MEATUS)

Eczema of the concha and the external meatus is of frequent occurrence in childhood; it is a tormenting and sometimes very obstinate disease. Usually it is associated with eczema of the head or face and seldom occurs independently. In other cases, where the patient suffers from eczema at other parts of the body, especially the lids, anus, umbilicus, olecranon, popliteal space, etc., the auditory meatus participates in the affection. Auricular eczema is either acute or chronic, according to the duration of the affection, and clinically we distinguish between vesicles, papules, moisture, scales, or crusts of eczema vesiculosum, papulosum, madidans, squamosum, crustosum, etc.

Furthermore, otitis externa eczematosa develops from neglected infectious purulent diseases of the external ear or middle ear. Every case of auricular furuncule or middle-ear suppuration may finally lead to eczematosis of the external ear. Canals invested with very vulnerable thin integument rather tend to squamous eczema, while a thick integument with plenty of fat and sebaceous glands tends to eczema madidans. Artificial eczema of the external ear may occur from scratching with hard, rough, or unclean articles to stop the itching.

Toxic eczema is sometimes observed after the use of iodoform or isoform in the treatment of the ear. Again, thermic or chemical irritation, arising from the use of volatile oils or strong-smelling substances, such as chloroform, camphor, Peru balsam, thymol, etc., may cause acute eczema of the external duct.

Eczema may also be the result of trauma caused by careless or exaggerated tamponade of the external meatus and impacted foreign bodies which irritate the skin. Persistent maceration of the integument resulting from continuous instillations in otitis media or from the swelling of foreign bodies (germinating beans) may develop into acute, eczematous inflammation of the external auditory duct with stormy symptoms.

Auricular eczema may also occur without any particular local cause in under-nourished, enfeebled individuals. This is liable to occur in rhachitis, chronic pulmonary tuberculosis, marasmus, anæmia, diabetes and untreated syphilis. Dyspeptic children frequently suffer from eczema. Nutrition plays an important part in infancy, eczema being far oftener observed in artificially fed, atrophic infants than in well-nourished, breast-fed children, although over-nutrition may likewise be a causative factor. Intertrigo, which is usually found where two skin surfaces are in contact, may occur behind the ear along the border of insertion of the concha, especially in very fat infants and children.

Symptoms.—Otoscopic examination will usually reveal a medium

degree of swelling of the skin of the external ear and an accumulation of crusts which may completely cover the tympanum. This swelling may in moist eczema lead to complete occlusion of the lumen. Acute eczema of the concha often sets in with considerable œdema and hyperæmia, vesicles and papules with serous or purulent contents, resembling herpes. In chronic squamous eczema the integument of the auditory duct is extremely thin, atrophic, easily vulnerable, and covered with a layer of brittle crusts which are easily detached. Slight mechanical irritation is sufficient to cause a profuse lymphatic secretion in chronic eczema of the external meatus. Skin irritations of this kind consist in rubbing or scratching with hard objects, syringing, or careless wiping with cotton tips.

Eczema of the external auditory duct has a peculiar tendency to relapse. The duct remains normal for some time, but gradually there is an accumulation of crusts and scales, which eventually exercise pressure on the integument and change without any further cause the squamous into the moist form. The latter then very rapidly spreads to the surrounding skin of the meatus, which becomes brittle, forming furrows and crusts, easily bleeding fissures in cold weather, and acute eczema madidans of the face and head. Relapses of the latter form are especially to be apprehended in exostoses of the external duct.

The epidermal layer of the tympanic membrane is unchanged in most cases of eczema of the ear; in some there is swelling, maceration, or desquamation of the epidermis, with injection of the vessels of the manubrium. Application of irritative remedies may also lead to inflammatory manifestations of the tympanic membrane in the form of reddening, swelling, and suppuration; the glands in the auricular region are generally enlarged and sometimes painful. The upper superficial cervical lymph-glands are particularly involved, likewise those of the inner surface of the submaxillary bone, whereas the mastoid gland is relatively rarely enlarged.

Itching of the skin is a very troublesome symptom accompanying eczema. It arrests all desire to work, causes headache and lassitude, and diminishes attention. Thus, children with itching eczema of the auditory canal are often unable to attend to their lessons and are consequently considered inattentive or incapable of concentration. Various nervous manifestations may be caused in the course of time, such as twitching in the region of the facial and cervical musculature, psychic excitability, and, in rare cases, epileptic convulsions. In the chronic form there is often a thin, abundant, yellow or brownish-yellow, highly malodorous secretion.

The functional test reveals a slight obstacle to sound-conduction, if the osseous part is permeable and the lateral tympanic membrane is

not directly covered with crusts. Should, however, the entire duct, or even its deep parts, be covered with crusts, the hearing acuity will be considerably reduced; eczema madidans has the same effect if the secretion fills the osseous part and covers the tympanic membrane.

The diagnosis offers no difficulties. The differential diagnosis should take into consideration acute, phlegmonous, or purulent perichondritis, herpes, lupus, and erysipelas. Acute eczema sets in with painless swelling and reddening of the skin, and, with the exception of the itching, there are no subjective complaints. Phlegmonous or purulent perichondritis, however, causes pains which will not subside until the abscess is in the process of formation, but in that stage no confusion with eczema is possible any longer. In herpes, vesiculation is associated with continuous, severe pains. As for lupus, see p. 390. In erysipelas of the concha there is always much fever, while the temperature in eczema is normal except in pronounced eczema madidans, which is associated with considerable œdema of the concha and lymphatic glands and with slight elevation of the temperature. Besides, thorough and repeated examination will serve to establish the characteristic demarcation and migration of erysipelas.

Several days' observation may sometimes be necessary to decide the question as to whether eczema of the external duct is associated with an affection of the middle ear. In doubtful cases of this kind the external auditory duct is carefully cleansed and sterile gauze strips are inserted and pushed forward into the osseous part. In profuse secretion they are renewed every hour, otherwise 2–4 times daily. In this way it will be possible to survey and examine the tympanic membrane, even in the most obstinate cases, on the second or third day.

A valuable aid for clearing up the differential diagnosis is the hearing acuity after cleansing the external duct and freeing the tympanic membrane from crusts or scales. A thin speculum is inserted to keep the duct permeable. Normal or slightly reduced acuity under these conditions would exclude a simultaneous affection of the middle ear, while considerable reduction of the acuity (to from 7–13 feet C.) would indicate its involvement.

Treatment.—In the presence of a demonstrable special cause, therapy and treatment should be adapted to the conditions, and the removal of the causative factor will often cause the eczema to disappear. Thus, in middle-ear suppurations care should be taken to provide for adequate drainage. In chronic suppuration of the middle ear, with caries of the temporal bone, radical operation is required to arrest the eczema. Should eczema develop in the course of middle-ear treatment, it would point to want of cleanliness or to the employment of unsuitable antiseptics which are not tolerated. Such an eczema will disappear

with sufficient attention to antiseptic rules and avoidance of strongly irritating agents (iodoform, isoform).

Eczema of the ear as part manifestation of general eczema or constitutional affection will only yield to local treatment in conjunction with general treatment.

In acute eczema madidans with considerable œdema of the concha or auditory duct, compresses and insertion of acetic alumina (1 : 6) or 1 per cent. lysol solution render good service, or in some cases compresses saturated with 95 per cent. alcohol, provided the latter is well borne and does not give rise to exacerbation of the subjective complaints. These compresses, however, are not allowed to remain *in situ* for more than a few hours. After removal, the ear is kept free for a few hours, followed by application of acetic alumina compresses. Alcohol insertions are not to be recommended in eczema of the auditory duct. Irrigations of the duct with water or washing the ear are contraindicated.

Eczema exhibiting slight moisture or scales should be treated with ointments in the form of compresses or inserted into the external duct. It is advisable to keep the ointment in a tube and squirt about 2–3 cm. on a sterile gauze strip. The ointment is enveloped in the gauze and inserted deep into the external duct. The advantage of this arrangement is that all crusts and scabs which the action of the ointment has loosened will adhere to the gauze and be removed with it on withdrawal.

The ointments to be recommended for eczema of the external auditory canal are boric (2 per cent.), zinc (2–5 per cent.), epicarin (2–5 per cent.), and resorcin (1 per cent.). As a base, ung. simplex, lanolin, or ung. emolliens is better than vaseline. Should there be considerable itching, 1–2 per cent. precipitate ointment is often very effective.

In simultaneous local irritation or furunculosis of the external duct an admixture of anæsthesin or cycloform is often advantageous.

The use of ung. diachylon simplex (sine ol. lavand. parat.) demands the greatest caution, as the unavoidable irritation of this preparation often leads to acute swelling with considerable pain. Squamous eczema calls for ung. emolliens; in chronic cases careful touching up with ½–5 per cent. silver nitrate and ear-baths with 1 per cent. potassa sulphurata are often attended with good results. Carbolin solutions or ointments (zinc oxide 4.0, carbolic acid 0.6, white vaseline 30.0), boralcohol, or tinctura rusci are tolerated only in rare cases.

Applications of desiccated powders (dry tampons of rice powder, calomel powder, vioform or airol powder) usually have but a temporary effect in lessening the subjective complaints. The exaggerated desiccation of the skin surface leads to friability of the epidermis and to relapses. On the other hand, mild powders, reducing the irritation (amylum and talcum preparations), have sometimes a very favorable effect in the

acute stages of conchial eczema. Local applications of yeast (mycodermin) or other yeast preparations (levulose, furunculin), used as powders, are likewise beneficial.

X-ray treatment is attended with surprisingly good, rapid, and permanent results in many cases of chronic squamous eczema.

I am in the habit of prescribing in all cases of eczema the internal administration of mycodermin (1 teaspoonful 1–4 times daily) and also arsenic in the form of Fowler's solution or "Levico" water. Improvement of the general condition, forced feeding, and a stay in a properly-selected country place are often very serviceable.

10. OTITIS EXTERNA FURUNCULOSA; OTITIS EXTERNA FOLLICULARIS (FURUNCLE OF THE AUDITORY DUCT)

Follicular inflammation of the auditory duct occurs from infection of the sebaceous glands. The first effect of the infectious germs seems to consist in agglutination of the superficial orifices of the sebaceous glands; as a consequence, the glandular secretion is retained in the glands, causing follicular inflammation and terminating with abscess formation owing to the action of the micro-organisms. The follicular inflammation always confines itself to the membranous part of the duct; since the osseous part does not contain any sebaceous glands and therefore cannot give rise to folliculitis. The inflammation of the membranous portion is either isolated or multiple, and contact of an abscess with the opposite wall may cause new inflammatory foci. Constitutional affections, under-nutrition, chronic eczema, and exostoses of the auditory duct will favor the occurrence of otitis externa follicularis. All affections of the auditory duct which produce itching may be the cause of folliculitis and infection of the sebaceous glands from patients violently rubbing and scratching themselves, thereby producing excoriations.

Symptoms.—The principal subjective symptom consists in considerable local pain, which may lead to sleeplessness. Since the anterior wall of the auditory canal participates in the movements of the maxillary articulation, patients will experience pain when speaking or masticating. Alcoholic beverages are apt to aggravate the pain considerably. The temperature is usually normal and only in exceptional cases slightly elevated.

Examination.—In introducing the ear-speculum, the ear should not be subjected to much pulling. If superficial inspection discloses considerable swelling and constriction of the duct, a thin funnel is selected, which is greased with a volatile oil to facilitate insertion. In the beginning of the affection it is only exceptionally possible to say what part of the wall is infected, as all the walls usually participate in the swelling. In advanced cases it may be possible at times to locate

the furuncle with the otoscope, without using the funnel. In deep furuncles the œdema of the duct will spread to the base and vicinity of the concha. In this way it is by no means rare for the œdematous swelling to involve the skin of the mastoid process (Figs. 64*a* and 64*b*), the parotid region, and in rare cases the inferior eyelid and the upper neck. In the latter case the mobility of the head and neck is interfered with, and a pathological oblique position of the head may be the result. In other cases furunculosis of the external duct may set in with a phlegmonous swelling of the base of the ear and the vicinity of the concha.

Fig. 64*a*.

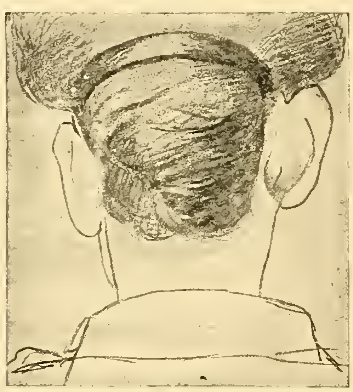
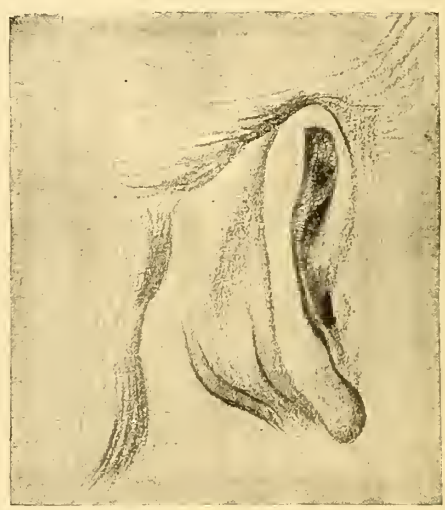


Fig. 64*b*.



Œdema of the mastoid region with painless swelling, in furunculosis of the right external auditory duct, in a girl thirteen years old. The concha of the affected side is entirely deflected and raised above the level of the healthy ear. The abnormal position of the concha can be most distinctly observed from the posterior aspect.

Course.—The course of otitis externa furunculosa is always favorable. In some cases it may be protracted, from the fact that new abscesses form by contact and new inflammatory foci develop. Failing competent treatment, extensive granulations may occur, which rapidly increase in size, filling out completely the entire external duct.

Diagnosis.—Generally speaking, the diagnosis of furuncle of the auditory duct presents no difficulties. The furuncle may be directly visible, and the healthy condition of the tympanic membrane may be established by the otoscope. Unless there are other affections of the ear, the acuity will be found normal in the test with the funnel inserted for the purpose of keeping the passage clear.

If the deep parts of the duct and tympanic membrane cannot be otoscopically inspected at the first examination, or if the duct is occluded by granulations, careful tamponade with xeroform gauze and removal of pus and crusts will clear the way. Granulomata may have to be

removed by the sharp spoon or snare before a decision is possible. Œdematous swelling of the ear caused by the furuncle will usually lead to œdematous swelling of the soft covers of the mastoid process.

Treatment.—Conservative treatment is indicated in the initial stages of otitis externa, as long as the auditory duct remains diffusely œdematous and the place of the future abscess is not yet discernible. Instillations and compresses of warm acetic alumina (1 : 6) or 1 per cent. lysol solutions have a very beneficial effect. Exaggerated or unpleasant pressure should be avoided when inserting the gauze tampons. Abscess formation should be stimulated by application of heat (instillation of hot liq. Burrowii, or carbolglycerin, or hot compresses upon the affected ear). As soon as perforation has taken place, the aspirating globe may be tentatively employed. Treatment with specific vaccine is also to be considered in the case of nurslings. The food should consist of fluids, and patients should speak as little as possible. All movements causing congestion of blood to the head are to be avoided. Easy evacuations are essential.

As soon as the place of the abscess formation has been definitely determined by color and swelling, it should be incised with a narrow-pointed scalpel, which is introduced into the external duct with its cutting edge over the furuncle. As the knife is withdrawn, the furuncle is longitudinally incised and a moist compress applied. This small operation is best carried out under ethyl chloride anæsthesia.

In local anæsthesia with ethyl chloride, great care should be taken to see that the tampon is inserted to a sufficient depth into the osseous canal. Anæsthesia by injection is not to be recommended.

In abundant granulation following perforated abscess, insertion and frequent renewal (6–8 times daily) of antiseptic gauze strips (xeroform, vioform, etc.) will often effect spontaneous involution. Granulomata completely filling the duct should be removed by the curette or snare.

Habitual occurrence of furunculosis is sometimes favorably influenced by insufflation of mycodermin into the external meatus and simultaneous internal administration of the same remedy (1 teaspoonful 1–4 times daily), preferably together with Fowler's solution, to prevent relapses. The ear should not be washed with water for a long time. A 3 per cent. borlanolinvaseline ointment is applied to the integument of the duct once or twice daily.

Repeated attacks of furunculosis yield most promptly and remain cured more often by the use of autogenous vaccine than by any other form of treatment. When, however, it is impossible to get such a vaccine, the mixed staphylococcus does the most good.

IX. AFFECTIONS OF THE MIDDLE EAR

I. TRAUMATIC INJURIES OF THE TYMPANIC MEMBRANE

The traumatic injuries of the tympanic membrane consist in lesions of the epidermal layer and mucosa and traumatic perforation. The epidermal layer may be injured during instrumental examination. Foreign bodies which have advanced as far as the tympanic membrane give rise to circumscribed lesions of the epidermal layer, which may become softened and macerated by the pathological process, exposing the tympanic membrane to perforation from injections. The mucosa may be injured by a bougie being pushed too far into the middle ear or by being bent in the operation. These two kinds of injuries, however, are in practice of far less significance than tearing or rupture.

Rupture may be direct or indirect. In direct rupture the tympanic membrane is destroyed by a solid or fluid substance advancing against the object of destruction. Indirect rupture is caused by condensation of air in the external auditory canal or middle ear, overtaxing the elasticity of the tympanic membrane. Indirect ruptures are far more frequent.

1. DIRECT RUPTURE OF THE TYMPANIC MEMBRANE

Direct rupture of the tympanic membrane is caused by perforation through pointed articles which have gained access to the external meatus, such as matches, toothpicks, lead-pencils, hair-pins, pen-holders, straws, peduncles of flowers or plants, wire, etc. Self-introduced articles rarely lead to rupture, for the reason that the osseous duct is very sensitive and even slight pressure gives rise to a very disagreeable sensation. Thus, self-induced rupture could occur only in hypæsthesia of the external duct and tympanic membrane, in very torpid individuals, or in extremely tormenting itching. Otherwise, rupture will occur by an article, lodged in the external duct, being accidentally propelled forward by a blow. Another danger is that fragile articles which have found their way into the tympanic cavity may break from the force of the blow. In such a case the foreign body can not be seen at all in the otoscopic picture, or the broken end will protrude through the rupture into the external duct.

However, all foreign bodies which have advanced beyond the isthmus into the osseous duct may cause traumatic injuries and rupture of the tympanic membrane. The mere penetration of water may cause traumatic rupture, especially when the resistance of the tympanic membrane has been impaired by catarrhal affections of the middle ear,

or when very thin circumscribed scars of the tympanic membrane are torn by a jet of water. Ruptures caused by falling or jumping into the water head foremost are nearly always of the indirect variety.

Direct rupture may also lead to secondary injuries, such as acute swelling of the tympanic mucosa, hemorrhage, or otitis media, when the fluid has penetrated into the middle ear.

In direct rupture the auditory duct is sometimes full of blood, and traumatic injuries of the integument may render an otoscopic examination difficult or impossible at the first sitting.

2. INDIRECT RUPTURES OF THE TYMPANIC MEMBRANE

occur from sudden condensation or attenuation of the air within the duct. The most frequent cause is a blow on the ear with the flat hand, not necessarily violent. The conditions most favoring this result are impaired resistance of the tympanic membrane and complete occlusion of the air within the duct at the moment of the blow, preventing the lateral escape of the air contained in the concavities of the concha and the external duct.

The next frequent cause of indirect rupture is a fall into the water, especially an unsuccessful head jump in the swimming bath, the water being liable to penetrate into the tympanic cavity and cause serous or purulent otitis media. Tender and thin scars of the tympanic membrane are likewise liable to be ruptured from forcible penetration of air. Explosions, detonations, falling from high altitudes, may cause rupture through concussion and air pressure, but these ruptures would be better classified under air concussions.

Symptoms.—Indirect ruptures may be without symptoms, especially when cicatrization has impaired the resistance of the tympanic membrane and the hearing acuity has not been normal before the rupture. The pain experienced in rupture of a normal tympanic membrane may be entirely absent in the destruction of a tender scar. Similarly, the slight reduction in hearing acuity may not be noticed in previously abnormal conditions, slight impairment of hearing being perceived only in injuries to a completely normal ear. The greater the functional injury before the trauma, the greater is the possibility of a further deterioration escaping the attention of the patient.

In most cases the occurrence of rupture is perceived as a short stabbing pain, often associated with a subjective noise, such as the perception of a high note or considerable tinnitus. More or less pain may be felt for several hours or days afterward, or there may be headache and vertigo. Occasionally, a tormenting sensation of fulness in the ear, numbness, headache, and nausea may persist for some time.

It is in the nature of the accident that patients are apt to exaggerate

the complaint, notably in cases of forensic importance. Thus, a previous affection of the ear is said to have become much worse, the hearing distance reduced, profuse ulceration is stated to have set in again in chronic ulceration of the middle ear which is previously supposed to have been more or less arrested.

In the presence of simultaneous injuries to the external ear, there will of course be regional symptoms in addition to those of rupture. In suffusions, tears, and contusions of the concha, or injury to the entire facial hemisphere, the real rupture symptoms will be quite subordinate to the severer symptoms of the totality of the injuries.

Otoscopic Findings.—In fresh cases there is either a slit-like, radial, or irregular fissure in the tympanic membrane (Fig. 65). The edges of the rupture are often irregular, fringed, or there is an angular tear with the flap folded upon the tympanic cavity or the external duct.

Fig. 65.

1 2

Traumatic rupture of the tympanic membrane after a blow on the ear. 1. Slit-like radial rupture of the left tympanic membrane; three hours after injury. 2. Circular rupture, larger than a hemp-seed, of a thin, atrophic right tympanic membrane; five days after injury.

The edges are tinged with blood, while the rest of the mucous membrane is either unchanged or infiltrated with punctiform hemorrhages. The rupture is sometimes followed by extravasation of blood through the external duct, the source of which can be detected at the lateral surface of the tympanic membrane. The fundus of the rupture in indirect cases is formed by the unchanged, medial tympanic wall, the yellow bone color of which is in marked contrast to the red margin of the perforation. Rupture of the two anterior quadrants is more frequent than those of the two posterior ones. In ruptures occurring on the otoscopic level of the stapes, window of the labyrinth, chorda tympani, etc., these parts will of course be seen through the rupture, and present the characteristic optic displacement, upon the examiner moving his head, as is observed in parallactic displacements of the eye.

Examination.—First establish the otoscopic findings. If the inspection of the tympanic membrane is impeded by a ceruminal embolus, crusts, blood coagula, etc., these obstacles may be carefully removed with sterile forceps, provided there is no suspicion of a fracture or fissure of the base of the skull. In the presence of such a suspicion, all manipulations of the external duct are to be deprecated in fresh cases. Syringing is absolutely contraindicated.

Valsalva's test will serve to demonstrate the characteristic rupture noise. In all cases where the tube is freely permeable and the ear has been normal before the injury, the air will escape through the rupture with a deep breathing noise.

If the tube was pathologically affected and is not permeable for the air current in a pharyngo-tympanal direction, the so-called reversed arrangement after Politzer is employed. By means of Siegle's funnel or Gellé's bulb, weak compressions are made in the external duct, the air escaping into the pharynx. The noise hereby produced can be distinctly perceived through the otoscope introduced into the nares, with patient's mouth kept open. A very convenient and convincing test is Kugel's. The otoscope—or, preferably, a rubber tube, 15 to 20 cm. long—is conducted into a glass of water, and, on application of Valsalva's test, air bubbles will be seen to escape through the water. The noise is in many cases audible at a great distance; in others it is so weak that it can be perceived only through the otoscope.

The functional test of the auditory duct reveals a slight obstacle to sound-conduction in uncomplicated cases, the rupture having caused slight reduction of the hearing acuity. The functional examination of the labyrinth is extremely important and should be made in all cases of traumatic injury of the ear. In cases of uncomplicated traumatic rupture the tympanic membrane can, of course, present no symptoms, being perfectly normal.

In complicated cases there will be spontaneous nystagmus and labyrinthine vertigo, or even equilibrial disturbance and vomiting.

Diagnosis.—If the history warrants and the otoscopic findings are distinct, the diagnosis of rupture offers no difficulties, especially if the rupture noise can be physically demonstrated. The irregular, sanguineously diffused margins in fresh cases are characteristic of traumatic rupture. In older cases the rupture may have assumed a circular shape with a white margin (corresponding to the young, new-growing epidermal layer), and the blood coagula may have been resorbed. This will render the differential diagnosis between traumatic and pathologic perforation of the tympanic membrane difficult; with incomplete history and in the absence of signs of healed old catarrh or purulent inflammation, the differentiation may be impossible. Furthermore, the place of rupture and coagula may change position, being shifted from the centre to the edge of the membrane.

Treatment of uncomplicated rupture of the tympanic membrane is confined to protection from injurious extraneous influence. The auditory meatus is closed with gauze, all manipulations are contraindicated, including, of course, every kind of irrigation or instillation.

Course.—In uncomplicated cases the gap will gradually close and the destroyed part of the tympanic membrane will be restored. In small circular ruptures up to the size of a hemp-seed, the membrane will be completely regenerated, so that the perforation will be perfectly closed by tympanic tissue, provided the membrane has been normal

before rupture. After the healing process is completed, the original seat of the rupture can no longer be traced, as the newly-formed tissue does not differ in any way from the rest of the membrane.

In atrophic conditions of the tympanic membrane in the region of the rupture there is usually new-formation of an atrophic membrane, but cases occur in which the newly-formed part is less atrophic than the one destroyed. According to the extent of considerable traumatic destruction of the tympanic membrane, there is danger of an atrophic scar closing the gap after healing has taken place. In complicating inflammatory processes of the middle ear there is, aside from the danger of purulent otitis media, the possibility to be reckoned with that synechiæ will form during the healing process, or that a permanent gap will form after the margins have been invested with epithelium. In angular rupture the peripheral part of the resulting flap may become necrotic, but healing will take place after desquamation. Some authors recommend removal of the irregular margins, but this is not only superfluous but even dangerous, since all instrumental manipulations of the ruptured tympanic membrane or the exposed mucosa of the middle ear may lead to otitis media.

The time required for healing depends upon the size of the gap and individual conditions. Ruptures from a millet-seed to a hemp-seed in size heal in 4–6 weeks. After the gap has healed the former hearing acuity, which is never much impaired in uncomplicated cases, will be fully restored, and a previously normal ear will again become perfectly normal. Complicated cases may end in permanent impairment of hearing, which, however, is not attributable to the rupture, but to the complication (acute catarrh of the middle ear, purulent otitis media, concussion of the lobe) and its sequelæ.

It may be mentioned that uncomplicated ruptures of the tympanic membrane, aside from the time required for healing, are to be regarded as slight injuries in a forensic respect.

II. INFLAMMATORY INFECTIONS OF THE TYMPANIC MEMBRANE

1. ACUTE INFLAMMATIONS OF THE TYMPANIC MEMBRANE (MYRINGITIS ACUTA)

Etiology and Occurrence.—Acute inflammation of the tympanic membrane is a frequent affection in childhood. It may occur as a primary inflammation without any known cause or after catching cold; as a rule, however, it is the forerunner of acute otitis media. It may, however, also be caused by direct or indirect traumatic injury of the external auditory canal or in connection with the removal of foreign bodies. Furthermore, all traumas which cause rupture of the tympanic membrane may cause its inflammation.

The anatomical changes consist in hyperæmia and œdema of the membrane, sometimes with formation of cysts of the lateral surface (exudative sacs). The exudate usually harbors demonstrable pathologic micro-organisms; other times it is sterile. Acute myringitis occurs either in one or both ears.

Symptoms.—There are stinging pains in the ear, which, however, do not attain to great severity and are sometimes merely indicated by the organic sensation of the tympanum. Hearing acuity is slightly diminished and the temperature is normal. Otoscopic examination always shows acute hyperæmia, sometimes also swelling, both in a considerably varying degree. Thus, in some cases the membrane appears unchanged with the exception of a radial vascular injection, in others there is diffuse hyperæmia; the line of the manubrium is either blurred or quite invisible, due to œdema. The cysts, if visible, are small and thin-walled, varying in size from a millet-seed to a small pea. They contain serum which is either limpid or blood-tinged; if purulent, which is rarely the case, they are yellow and opaque. With yellow and clear serum, the otoscopic picture resembles in color and lustre that of secretory catarrh of the middle ear. The lateral wall of the tympanic cysts is formed by the epidermal layer of the tympanic membrane; the medial wall, by the remaining layers of the tympanic membrane (substantia propria and mucosal layer). There is no communication between the cysts or their contents and the tympanic cavity. Cyst formation is more frequently observed in the posterosuperior quadrant than in the other parts of the membrane.

Functional examination demonstrates in all cases an uncomplicated slight obstacle to sound-conduction.

The diagnosis of acute inflammation of the tympanic membrane is based upon the otoscopic findings. To make a differential diagnosis from simple acute (serous) inflammation of the middle ear the slight degree of functional disturbance, slight pain, and normal temperature have to be considered. There can be no diagnostic difficulties, as simple inflammation of the middle ear always commences with much pain and, at least during the first days, with considerable impairment of hearing; there is also elevated temperature in most cases.

Course.—The course of the affection is favorable in many cases, the symptoms completely disappearing in a few days, and normal hearing returning within a week, with complete restoration of the normal picture of the membrane. In other cases, however, the affection is the forerunner of acute simple or purulent inflammation of the middle ear. The change from an inflammation of the tympanic membrane to one of the middle ear is plain from the rapid reduction of the hearing acuity, the increase of subjective complaints, and the elevated temperature.

It is a noteworthy fact that, where inflammation of the middle ear is the result of inflammatory changes of the nasopharyngeal tract, an habitual tendency to relapses may persist, causing—with every coryza, for instance—inflammation of the tympanic membrane, with or without formation of exudative sacs. This may finally lead to atrophy of the tympanic membrane.

The treatment of acute myringitis is identical with that of acute simple (serous) inflammation of the middle ear, for which see p. 156.

2. SUBACUTE AND CHRONIC INFLAMMATION OF THE TYMPANIC MEMBRANE (MYRINGITIS SUBACUTA AND CHRONICA)

Light degrees of subacute and chronic myringitis are of very frequent occurrence in childhood, and may develop from the acute stage owing to adenoid vegetations or where the nasopharyngeal tract has been disturbed by other changes. The subjective complaints disappear with the acute stage, but slight impairment of hearing and injection of the tympanic blood-vessels will persist for some time. The dull, gray-red, retracted tympanic membrane of childhood is almost pathognomonic for the presence of adenoids. The findings in subacute and chronic myringitis are not always constant, as temporary improvement may occur at any time, during which the picture of the tympanic membrane acquires an approximately normal appearance. Should the causative factor persist, there may be periodical aggravations, during which hyperæmia is increased and serous or purulent otitis media develops.

The ulcerative form of chronic inflammation of the tympanic membrane (myringitis chronica ulcerosa) is a rare affection, and consists of ulceration and chronic purulent inflammation of the tympanic membrane, the ulcerations occurring on the middle as well as the lateral surfaces. Otoscopically, they are only visible at the lateral surface.

Myringitis ulcerosa occurs during periods of abatement of chronic ulcerations of the middle ear, or as an independent affection in debilitated, anæmic, or tuberculous individuals without any known cause.

The diagnosis is made on the otoscopic findings of the ulcerations.

The course is unfavorable in many cases, as there is gradual development of suppuration of the middle ear. In other cases, however, it is favorable, and a complete cure will result under appropriate general and local treatment.

The treatment of myringitis ulcerosa consists in cauterizing the ulcerated surface with chromic acid pearl, ferum sesquichloratum, or trichloracetic acid. Improvement of the general condition of nutrition by forced feeding, sojourn in the country or at the seaside, and sea baths, etc., is of importance. With an increase in weight there is often spontaneous involution after a case has proved refractory to all local

treatment. Where chronic meningitis is a part manifestation of suppuration of the middle ear, only the latter calls for treatment.

III. CATARRHAL AFFECTIONS OF THE MIDDLE EAR

Catarrhal affections of the mucosa of the middle ear are associated with swelling of the mucosa and secretion of an exudate which, in fresh cases, is serous, limpid, and light yellow; in chronic cases, purulent in various degrees. Microscopically, the exudate contains a small number of mononuclear leucocytes and mast-cells, as well as various bacteria, mostly degenerated. Clinically, the exudate is of very little importance in many cases; in chronic cases it is entirely absent. All middle-ear catarrhs in which there is considerable exudation are comprised in the generic name of "exudative middle-ear catarrh," as differentiated from all others (Politzer).

Etiology.—Middle-ear catarrhs are primarily traceable to acute or chronic changes of the nasopharyngeal tract in the majority of cases. The principal etiological factor in children is enlargement of the faucial tonsil (adenoid vegetations). Of further etiological importance are all so-called colds. All affections associated with hyperæmia and catarrhal changes of the nasopharyngeal tract may lead to catarrhal involvement of the middle ear in the acute as well as later in the chronic stage. Disturbed ventilation of the tympanic cavity always leads sooner or later to catarrhal affection of the middle ear. If the pharyngeal opening of the tube has become impassable from swelling of the mucosa or is constricted or occluded by a tumor, the physiological function of the tube is interfered with, leading to disease of the middle ear. Furthermore, catarrh of the middle ear is a typical concomitant manifestation of the etiological primary affection in disturbed motility of the tubal musculature, paralysis of the velum palatum, uranoschisis (cleft palate), nasopharyngeal tumor, tumor of the superior maxilla, syphilis, nasal tuberculosis, rhinoscleroma, etc. It is only rarely that catarrh of the middle ear follows traumatic injury of the tympanic membrane, tympanic cavity, or Eustachian tube. Noxious fumes or gases may indirectly cause catarrhal affection of the middle ear; the fumes first produce catarrh of the nasopharyngeal mucosa, from which the inflammation spreads to the tubal mucosa and thence to the middle ear. Finally, catarrh of the middle ear may be a deuteropathic manifestation of healed purulent otitis media.

Operations of the nasopharyngeal tract, hard or soft palate may be the cause of catarrhal affections of the middle ear due to the reactive swelling of the nasopharyngeal mucosa.

1. Acute catarrh of the tube generally develops at the climax of acute colds (coryza), and is often observed in children as an early

stage of acute infections. The otoscopic findings (Fig. 66) show no changes save a retraction of the tympanic membrane. The hearing acuity is not materially reduced; sometimes there is a feeling of fulness in the ear, which may be accompanied by subjective noises. As the inflammatory changes abate in the nasopharyngeal space, the tubal catarrh will heal within a few days without any local treatment.

2. Acute catarrh of the tympanum is rather rare and exclusively due to the influence of trauma in the auditory canal. It is occasioned by the penetration of hot steam into the latter (explosions, etc.); furthermore, catarrh restricted to the tympanic cavity may occur in cases of rupture of the tympanic membrane, if a foreign body advances into the middle ear through the opening of the rupture (direct injury from toothpicks or matches, indirect injury from penetration of water). A more frequent sequel to this injury, however, is purulent otitis media.

Fig. 66.

Considerable retraction with pronounced protrusion of the posterior, superior, and anterior folds of the tympanic membrane.

The course is usually favorable. Œdema and hyperæmia of the mucosa of the middle ear, which can be well observed through the rupture, rapidly abate, and the impaired hearing distance soon returns to normal. As to therapeutic measures, it is sufficient to cleanse the external duct with dry cotton tips or squeezed-out peroxide swabs, closing the same with antiseptic or sterile gauze strips.

3. Acute and subacute catarrh of the middle ear (secretory middle-ear catarrh, catarrhus recens) is usually a sequel to colds, or rather to the catarrh of the upper air-passages resulting therefrom (particularly of the nose and nasopharynx).

The principal symptom of exudative middle-ear catarrh consists in a more or less pronounced reduction of the hearing acuity (down to 3½–10 feet C.), which may develop in the course of a few hours. There may also be subjective noises (falling of drops), unless the middle-ear spaces are completely filled with exudate; sometimes there is a sensation of fluid in the ear, participating in the movements of the head and body, and causing a characteristic splashing noise.

There are still to be mentioned sensation of fulness and pressure in the ear, numbness in the head, and resonance of patient's own voice. Painful sensations are not rare in middle-ear catarrh with acute onset; they are not caused by the catarrhal affection itself, but by the simultaneous acute inflammatory changes of the nasopharyngeal tract, the latter being also exclusively responsible for any possible elevation of temperature.

Otoscopic examination shows normal position or slight retraction of the tympanic membrane. The lustre of the membrane is increased,

the light reflex is more intense, giving the impression as if the membrane were varnished, the yellow exudate imparting its color to the membrane. If the entire mesotympanum is filled with exudate, the whole of the tympanic membrane is tinged yellow (Plate VIII, Fig. 1). When there is any residue of air in the tympanic cavity, the lower, yellow part of the membrane is sharply demarcated against the gray upper part through the meniscus formed by the fluid (Plate VIII, Fig. 2). This line of demarcation closely resembles a hair in the otoscopic picture and is always distinctly visible. Changing the position of the head causes the demarcation line to change also, the fluid gravitating toward the lowest point of the tympanic cavity. This line is in many cases more or less straight, in others simply curved or undulated. It extends over the entire width of the membrane or, as the exudate increases, it is confined to the anterior or posterior half of the membrane. The increased lustre of the tympanic membrane is occasioned by osmosis, and from the same cause the manubrium appears thin and narrow. In the normal otoscopic picture the strip denoting the latter represents not only the bone, but also the connective tissue encircling it (Fig. 25, *c*, Plate VIII, Fig. 1). For this reason the manubrium appears thicker in the normal otoscopic picture than in the skeleton. If now the tympanic membrane is humidified through osmosis, the rest of the membrane and the connective tissue encircling the manubrium become transparent, and nothing but the bone is visible in the shape of a sharply demarcated yellow strip.

The functional test shows all the signs of an obstacle to sound-conduction in uncomplicated cases.

Only the smallest part of the exudate comes from the catarrhal mucosa of the tympanic membrane, the greater portion emanating from the aspirated secretion of the mucous glands of the tube, as the catarrh sets in with occlusion of the tube. The tympanic cavity is no longer ventilated and the air contained in it is partly resorbed. In this way the over-pressure of the outer air causes passive retraction of the tympanic membrane. The tympanic cavity, containing rarefied air or none at all, aspirates the secretion of the tubal glands, which, besides, is increased from catarrh. As this secretion cannot escape through the pharynx, it wanders upward into the tympanic cavity. This form of exudation is called passive. Active exudation, in which the exudate is under considerable pressure in the middle-ear spaces, always causes the tympanic membrane to bulge out. Active exudation of this kind always occurs in serous as well as in purulent otitis media.

The diagnosis offers no difficulties. Doubtful cases should always be examined in daylight, as the yellow or yellowish-brown coloration of the tympanic membrane is then particularly distinct.

Treatment.—There should be no local treatment whatever in the first days of illness. The patient should remain in bed if the temperature is elevated. In the evening he is given hot lemonade and an infusion of tilia flowers and aspirin (children 0.15–0.5 Gm., according to age; adults 1–2 Gm.). Violent perspiration often causes spontaneous improvement of the ear trouble. If the body temperature is normal and the acute catarrhal or inflammatory manifestations of the nose and throat have subsided, applications of the Politzer air-douche, twice or three times weekly, may rapidly improve the hearing acuity and completely cure the affection in 1–2 weeks. In applying the air-douche, the longitudinal axis of the rubber tube is placed in a vertical direction. The patient keeps a draught of water in his mouth; his head is inclined toward the healthy side and turned toward the affected side, and in this position the air is injected. The air ascends in the middle ear-space and the exudate is displaced by the air, escaping downward through the vertical tube.

Insufflation of air serves instantaneously to atomize, aërize, or displace the entire exudate, which will either be pushed into the antrum and cavities of the mastoid, or else flow off through the tube. However, exudate will collect again in the tympanum in the next few hours. Examination of the patient 24 hours after insufflation of air will still show demonstrable exudate in cases taking a favorable course, but in reduced quantities. Where previously the entire tympanum was filled with exudate, there is now a line of demarcation; where there was a border line before, it now occupies a lower position.

Where the conservative treatment fails, there are always renewed accumulations of exudate, and the only remaining remedy is paracentesis. The aperture thereby made admits air into the tympanic cavity, the hearing distance is at once increased, and in many cases there is spontaneous evacuation of the exudate. Should there be no spontaneous evacuation, the exudate may be forced out by insufflation of air either at once or twenty-four hours after paracentesis.

As a matter of course, all aseptic precautions should be observed in doing a paracentesis; nevertheless, it may not always be possible in acute cases to prevent postoperative purulent inflammation of the middle ear. The mucopurulent secretion usually persists for 1–2 weeks, but is finally completely arrested. The danger of postoperative purulent otitis media is particularly great where there is a transition form of exudative catarrh into acute serous inflammation of the middle ear (Plate VIII, Fig. 6).

In the **differential diagnosis,** both for children and adults, otosclerosis should be considered. In the initial stage or at the climax of atypical otosclerosis there are sometimes true exudative processes of the

middle ear. As a matter of course, the prognosis of these exudates is unfavorable in view of the fact that otosclerosis is the primary affection. The suspicion of otosclerosis, hidden behind the picture of exudative catarrh, is justified if there was impaired hearing before the middle ear became affected or if insufflation of air for test purposes did not lead to any material improvement of the hearing acuity.

4. Chronic tubal catarrh is a rather rare affection, since chronic catarrhal affections of the tube very soon involve the entire middle ear in the majority of cases. Otoscopic examination shows considerable retraction of the otherwise unchanged tympanic membrane. The short process and the posterior tympanic fold protrude considerably; Shrapnell's membrane is much retracted. The otoscopic picture sometimes simulates perforation of Shrapnell's membrane, and it must be left for examination with Siegle's speculum to demonstrate the condition of the highly retracted membrane. The tube is either of normal width or constricted by swelling of the mucosa.

Hearing acuity is variable, patients hearing sometimes very well and at other times very imperfectly. There is numbness in the head, and there are usually deep, tormenting subjective noises. Catheterizing of the Eustachian tube causes crepitant râles; Politzer's tuning-fork test is negative.

Prognosis and treatment of chronic tubal catarrh are chiefly dependent upon the primary affection of the nasopharynx. The prognosis is favorable if the affection is caused by remediable changes, such as adenoid vegetations, hypertrophy of the nasal septum, pathological enlargement of the nostrils, or acute ulceration of the accessory cavities. When these obstacles are removed, the hearing distance becomes and remains normal after short treatment, consisting in Politzer's method of air insufflation, applied two or three times a week. On the other hand, permanent improvement is impossible if the tubal catarrh is caused by irreparable or chronic purulent changes in the nasopharyngeal space (malignant tumor, chronic ulceration of the accessory cavities, deformities or defects of the hard palate). In these cases the catheter can only free the patient for some time from an ear trouble which is often very grave. If the nose is impassable, the catheter will have to be introduced through the oral cavity. The considerable retraction of the tympanic membrane usually causes subjective hearing sensations from which patients suffer more than from impaired hearing. This condition can momentarily be relieved by the application of Delstanche's massage instrument, used as an aspirating apparatus.

A child suffering from chronic tubal catarrh can only with difficulty follow his school lessons, owing to the periodical or permanent impairment of hearing acuity, and will therefore be a backward pupil.

5. Chronic catarrh of the middle ear in early childhood is primarily traceable to chronic changes in the nose and nasopharyngeal space, and particularly to adenoid vegetations. The highly disturbed physiological function of the tube gradually leads to grave deuteropathic manifestations of the tympanic membrane and the mucosal fold of the tympanic cavity, the result being circumscribed or diffuse atrophy, considerable retraction of the tympanic membrane, and bulging of the posterior fold of the membrane (Fig. 67). The retraction may become so considerable that in the otoscopic picture the manubrium appears to be behind the tympanic fold and is therefore invisible.

Chronic catarrh of the middle ear in childhood undergoes many variations in the course of time. Under the influence of colds, coryza, infectious diseases, etc., it may lead to acute exacerbations and acute inflammatory irritations of the middle ear. In other cases chronic exudative catarrh or a chronic adhesive process may develop.

Fig. 67.

1 2 3

Tympanic membranes in catarrhal affections of the middle ear. 1. Healed middle-ear catarrh; kidney-shaped atrophy of the posterior, superior, and inferior quadrants. 2. Chronic middle-ear catarrh; central atrophy. 3. Chronic middle-ear catarrh; high-grade retraction; central atrophy.

The treatment should be energetic and commence with the nasopharyngeal tract. The air-passages should always be made permeable by operative interference, removing all obstacles. After one or two weeks' intermission there is sometimes spontaneous improvement of the catarrh and hearing acuity.

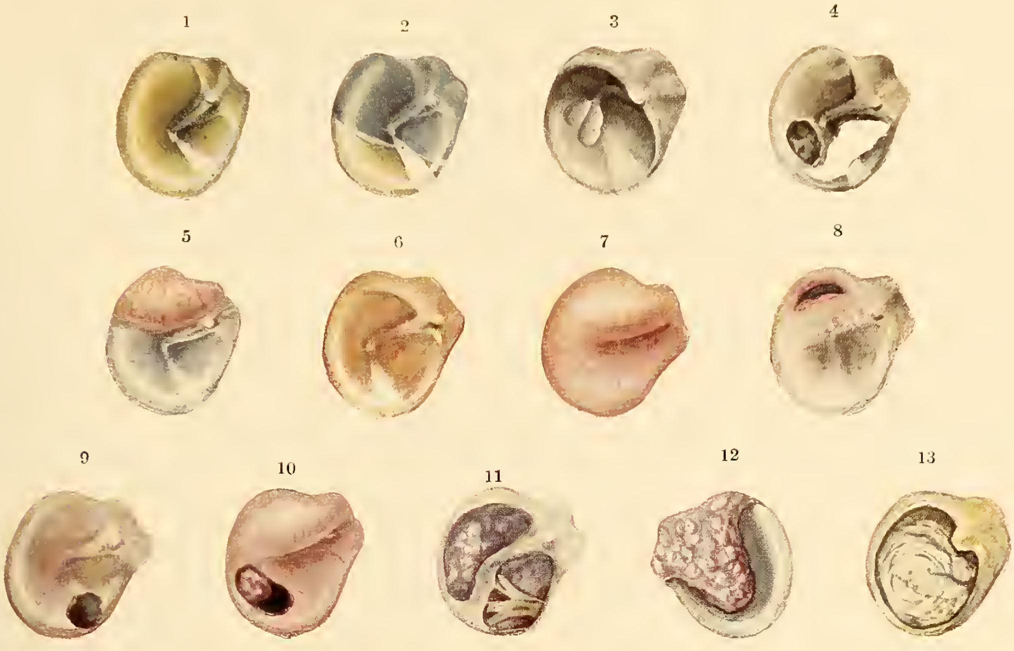
Gaertner's rhinometer is an excellent instrument for measuring the precise permeability of the nose for air currents.

Further improvement in the hearing acuity may then be effected by air insufflation two or three times a week and by aspiration with a bulb or Delstanche's massage instrument. Entirely normal hearing will result only in the rarest of cases. Valsalva's experiment should be rigorously forbidden, as it conduces to extend the atrophy in whatever degree it may be present and eventually cause traumatic rupture of the tympanic membrane.

The prognosis depends upon the functional findings. It is favorable if at the first examination equalization of air pressure by insufflation materially improves the hearing acuity. With only moderate improvement, a complete restoration of function cannot be expected, even when the therapeutic measures are strictly observed. In some cases indeed the gradual transition of simple chronic middle-ear catarrh into the more unfavorable chronic adhesive process cannot be arrested.

6. Chronic exudative middle-ear catarrh is either a continu-

PLATE VIII.



Pathological otoscopic findings of the tympanic membrane. Enlarged 2 : 1.

Explanation of Illustrations.

FIG. 1. Acute secretory catarrh of the middle ear: Tympanic membrane tinged wine-yellow or amber-yellow; lustre increased; line of manubrium narrow.

FIG. 2. Acute secretory catarrh of the middle ear: Lower part of the tympanic membrane tinged yellow, the upper part has the normal bluish-gray tint. The yellow part is demarcated upward by the meniscus of the fluid.

FIG. 3. Chronic adhesive process: Tympanic membrane considerably retracted, posterior and anterior folds of the tympanic membrane very distinct, membrana tensa atrophic, reflex well preserved; two central glistening scars. In this case the chronic adhesive process has developed as a sequel to repeated catarrhal affections of the middle ear.

FIG. 4. Chronic adhesive process after cured middle-ear suppuration: Preserved perforation of the posteroinferior quadrant; intermediary lime deposits anteroinferiorly; the posterior and superior parts of the tympanic membrane are diffusely atrophied.

FIG. 5. Acute epitympanic inflammation of the middle ear, with principal involvement of the antrum with protrusion and injection of the posterosuperior quadrant of the tympanic membrane.

FIG. 6. Transition form of secretory catarrh into acute serous inflammation: Frequently found in childhood in the presence of adenoid vegetations. The lustre of the tympanic membrane is preserved, the latter showing a yellowish-red coloration.

FIG. 7. Acute purulent otitis media with considerable protrusion of the entire tympanum; the line of the manubrium, which is indicated by a narrow furrow, cannot greatly participate in the protrusion.

FIG. 8. Chronic tuberculous suppuration of the antrum, with small, fissure-like perforation of the posterosuperior quadrant; the tympanic membrane is turbid and dull, and the line of the manubrium is blurred through being encircled by connective tissue.

FIG. 9. Chronic suppuration of the middle ear, with principal involvement of the tube and hypotympanum; perforation the size of a hemp-seed in the anteroinferior quadrant.

FIG. 10. Chronic suppuration of the middle ear with acute relapses: The chronic ulceration of the middle ear corresponds to the oval perforation of the posteroinferior quadrant and the granulation of the tympanic cavity. Part of the granulations of the promontory and the rest of the middle wall of the tympanic cavity are visible in the gap of perforation. In this case the chronic suppuration has led to the formation of a septum in the mesotympanum, so that the developing acute epitympanic otitis is associated with considerable bulging of the posterosuperior quadrant, which largely covers the line of the manubrium. The secretion accumulated behind it could not escape through the perforation and had to be evacuated by paracentesis in the posterosuperior quadrant.

FIG. 11. Chronic suppuration of the middle ear with extensive double perforation: The middle wall of the tympanic cavity is covered with granulations; in the anteroinferior quadrant in the area of the tympanic ostium of the tube there are three broad ligamental layers of connective tissue. In this case there were considerably reduced hearing acuity and irritative manifestations of the static labyrinth in the form of vertigo, equilibrial disturbance, and pathologically increased labyrinthine reflex excitability.

FIG. 12. Chronic suppuration of the attic and antrum with formation of a racemose granulation polyp.

FIG. 13. Chronic suppuration of the middle ear with destruction of the tympanic membrane: Concentrically striated cholesteatomeus plates are visible in the perforation.

ation of the acute condition or it arises from the fact that in chronic simple middle-ear catarrh the secretion of the tubal glands is aspirated into the tympanic cavity owing to air resorption in the latter.

The findings of the tympanic membrane are usually characteristic. To begin with, there are all the signs of chronic catarrh: retraction, protrusion of the posterior fold, striated or cloudy turbidity, cicatrization, atrophy, etc. To these signs are added the diagnostic symptoms of the tympanic exudate: increased lustre of the tympanic membrane (saturated membrane), narrow line of the manubrium, the characteristic yellow color, emanating from the exudate, at places where the tympanic membrane has retained its normal transparency or, owing to atrophy, is more transparent than usual. In the presence of widespread atrophy the various parts are sharply separated by protruding crests; each atrophied place is considerably retracted, injected, and has punctiform light reflexes.

The diagnosis presents no difficulties if the yellow coloration of the tympanic membrane extends over the entire or a considerable part of the field of vision. If, however, the tympanic membrane is thickened and, with the exception of a few atrophied places, shows gray or grayish-white discoloration or calcification, the yellow color is only noticeable at the small atrophied places or may be entirely absent, and then the diagnosis must be made on the strength of the increased lustre, osmosis of the tympanic membrane, and the narrow line of the manubrium. But even with this reduced number of signs the experienced physician will have no difficulty in arriving at a diagnosis.

Treatment consists in evacuation of the exudate by paracentesis. Spontaneous evacuation should be waited for before further interference, in order to prevent postoperative inflammatory irritation or suppuration. The auditory meatus is closed with sterile gauze strips. If there is no spontaneous evacuation after 24 hours, Politzer's air insufflation is applied, or, failing this, owing to extensive tubal changes, the exudate is conveyed to the external duct by means of the catheter. The exudate is always mucous, stringy, and sometimes consists of nothing but mucus, which, after air insufflation, presents itself like a colloid foreign body in the external duct through the puncture, where it can be removed with forceps. Placed in a formalin solution, this mucous lump soon resumes its natural shape, showing a distinct cast of the tympanic cavity, antrum, and tubal ostium.

The further treatment consists in two or three air-douches weekly under constant control of the hearing acuity. The puncture completely closes in from two to three weeks, after which treatment of the nasopharyngeal tract is proceeded with in order to avoid recurrence of the catarrh.

7. Subacute recurrent middle-ear catarrh is principally observed in children suffering from adenoid vegetations.

Acute inflammatory swelling of the enlarged faucial tonsil occurring in the course of a common cold or coryza will in these children immediately lead to all the symptoms of middle-ear catarrh. Recurrent middle-ear catarrh may sometimes be of the simple and sometimes of the exudative type, or of both together, in one and the same patient. Without proper treatment these relapses will increase in frequency and obstinacy. Examination in the intervals between two attacks may not reveal any particular enlargement of the faucial tonsil; nevertheless, its removal is imperative, lest the hearing acuity be permanently injured. Some of the relapses may be of the ulcerative type, and, if these children contract an acute infectious disease, there is considerable danger of grave middle-ear infections resulting.

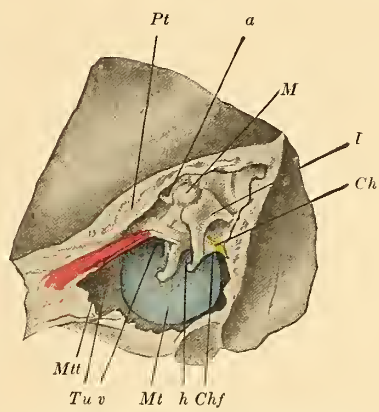
IV. THE CHRONIC ADHESIVE PROCESS

The designation of chronic adhesive process (chronic adhesive inflammation of the middle ear) comprises all those affections which represent the termination of catarrhal or purulent affections of the middle ear. At the present juncture it is proposed to discuss only those adhesive processes which result from catarrhal affections of the middle ear. In the majority of cases they are the final stages of chronic exudative catarrh in which the exudate has been gradually organized by connective-tissue formation.

There is, in the first place, thickening and stiffening of the ligaments and mucosal folds located in the tympanic cavity (Fig. 68). Superficial or more or less deep layers of connective tissue develop in the tube, which impede its mobility. The act of deglutition has no longer any effect upon the tube, or the tubal lumen becomes constricted with the result of grave tubal stenosis or stricture, which is most liable to appear at the transition of the membranous into the osseous tube. Folds and layers of connective tissue develop at the pharyngeal tubal ostium, which may cause its complete occlusion. The thickening and rigidity of the ligaments and folds reduce or arrest the mobility of the chain of auricular ossicles (Fig. 68). The corner of the labyrinthine window becomes replete with connective tissue and may finally be obliterated. In unfavorable cases the chronic adhesive process leads to complete obliteration of the middle ear by connective tissue (atresia); in other cases, to circumscribed connective-tissue proliferations of the middle ear, with the result of high-grade functional disturbances, which, as a rule, are irremediable. The tympanic membrane itself sometimes participates slightly in these changes, while in other cases it is considerably involved (Plate VIII, Fig. 3).

Diagnosis.—The possibility of a chronic adhesive process should be considered if examination with the aid of Siegle's speculum demonstrates reduced or inhibited mobility of the tympanic membrane. A chronic adhesive process may, however, also be present in cases in which the mobility of the membrane is well preserved, or even increased, owing to atrophic conditions, provided the examination shows that the malleus is fixed and does not participate in the movements of the tympanic membrane. The air-douche sometimes gives negative results in highly constricted tubes, the pathological occlusion being so firm that the air current is unable to penetrate it. Application of the catheter reveals in many cases a tube of normal width, in others more or less pronounced stenosis or stricture of the tube which can be topographically located with the aid of bougies.

Fig. 68.



Lateral wall of right tympanic cavity and membrane (*Mt*) in a chronic adhesive process. The typical mucosal folds are thickened and rigid, showing a system of pathological connective-tissue layers (*a*) in the epitympanum, encircling the head of the malleus and completely arresting the mobility of the latter. (Enlarged 3 : 2.) Child twelve years old. *Pt*, paries tegminis; *M*, malleus; *I*, incus; *Ch*, chorda tympani; *Chf*, fold of chorda; *h*, posterior pocket of tympanic membrane; *v*, anterior pocket of tympanic membrane; *Mtt*, musculus tensor tympani; *Tu*, tuba auditiva.

There is suspicion of a chronic adhesive process if in a case of apparent middle-ear catarrh neither air-douche nor catheter is able to improve the hearing acuity even slightly. The danger of gradual spontaneous transition of simple middle-ear catarrh into an adhesive process increases with age.

Constriction or occlusion of the pharyngeal tubal ostium by connective-tissue proliferations may render catheterization difficult or impossible.

The functional test shows a typical disturbance of sound-conduction, provided the labyrinth has remained in normal condition (see below). The only difference as compared to simple middle-ear catarrh is that the hearing acuity cannot be materially improved, if at all, by physical measures (air-douche, catheter, aspiration, pneumomassage).

Prognosis and Course.—There is danger in every pathological connective-tissue formation of the middle ear that it will spread to the labyrinth. At first a network of connective tissue develops, which is later filled up at the basal end of the vestibular part of the cochlea and followed by pathological ligament formation in all perilymphatic spaces, with consequent atrophy of the labyrinthine nerve-ends, nerve-branches, and the peripheral ganglia of the cochlear nerves. Should, therefore, the clinical functional test show the cochlea to be in any way affected, coupled with reduced perception of high sounds and reduction of the upper sound-limit, the prognosis is absolutely unfavorable.

The adhesive process with an unchanged labyrinth takes a variable course: benign cases experience more or less considerable and permanent improvement of the hearing acuity under timely and competent treatment. In other cases gradual deterioration cannot be arrested by any kind of treatment.

This difference in results depends upon what kinds of adhesions are formed in the tympanic cavity. Strong adhesions consisting of connective tissue offer a less favorable prognosis than cases in which even more extensive connective-tissue adhesions prevail which, however, are elastic and well studded with nuclei.

Treatment.—Treatment of the nasopharyngeal tract must in all cases precede that of the local ear affection, and the directions given for various forms of middle-ear catarrh in regard to nasopharyngeal therapy again hold good.

Local treatment in chronic adhesive processes of the ear spells failure, unless preceded by appropriate treatment of the nasopharyngeal tract.

The object of conservative local treatment is to improve the position of the tympanic membrane, remove any secretion that may be present, stretch, soften, or resorb the adhesions, and thereby improve the impaired mobility of the chain of auricular ossicles. To obtain the best practical results, local treatment should commence with that part of the middle ear where the changes are most pronounced.

The physical treatments consist in insufflation of air and catheterization, both of which may be combined with application of medicated vapors. Those most used are acetic ether and mixtures of acetic and chlorated ether (5.0 āā). Politzer recommends vapors of turpentine oil (oleum terebinthinæ 15.0, mentholi 1.0); Urbantschitsch advises camphorated ether (10 per cent.); Gomperz uses ammonia, for which he has devised a special apparatus. Application of vapors to the middle ear is in many cases attended with considerable and permanent improvement of the hearing acuity and a favorable effect upon the tiresome subjective noises.

Injections of medicated fluids through the catheter and the tube into the middle ear are intended to stretch, soften, and eventually resorb the adhesions. They are preferably made in the dorsal decubitus, the correct position of the catheter in the tubal aperture having been ascertained by a test insufflation of air. In the first sittings only a few drops are injected, the dose being gradually increased to 0.5 c.c. The fluid is propelled into the middle-ear spaces by subsequent air insufflation. The following injections have been recommended: Oleum vaselini sterilisatum, sodii bicarbonas (natr. bicarb. 0.5, aq. dest. 10.0, glycerini 2.0 : 8–10 drops), pilocarpinum hydrochloricum in a 1–2 per cent.

aqueous solution, potassium iodide (pot. iod. 1.0, aq. dest. 10.0), zincum oleinicum (zinc. olein. 0.3, ol. vasel. 30.0 : 8–10 gtt.), zincum oleinicum vasogenatum, and adrenalin (sol. adren. 1.0, glycerini, aq. dest. āā 15.0). Many authors recommend pilocarpin in the form of subcutaneous injections into the upper arm. A few drops of a 1 per cent. solution are injected with the utmost care, as even the slightest doses may produce toxic manifestations in intolerant patients (long-continued nausea and vomiting, collapse, weakening and continued perspiration). Latterly, fibrolysin has been applied both through the tube and subcutaneously in the mastoid region. Injections of fibrolysin into the tympanic cavity, however, may very easily lead to undesirable acute inflammatory changes of the middle ear.

Introduction of bougies into the tube through the catheter is indicated in tubal stenosis or stricture. The calibre of the bougies should gradually increase, commencing with the smallest. This treatment is instituted twice a week and continued for 4–6 weeks.

If an operation on the nasopharyngeal tract has been previously performed, treatment with catheter and bougies should be delayed until the wound has completely healed, which will generally be the case in three or four weeks. Otherwise there is danger, in acute inflammatory affections or in the presence of fresh traumas, that traumatic, serous or purulent otitis media may result. In properly selected cases the therapeutic success of the bougie treatment is excellent. However, in normally passable cases, in which there is simply catarrh of the tube to be dealt with (moist crepitant râles during catheterization), mere insufflation of air or catheterizing is the indicated treatment.

Locally irritating fluids (weak solutions of acetic alumina, silver nitrate, potassium chloride, lithia carbon.) cause acute inflammation of the middle ear which occasionally leads to remarkable improvement of the hearing distance. Nevertheless, this method is not to be recommended, since it may give rise to grave suppuration of the middle ear; besides, the improvement is not permanent, the hearing acuity being reduced to its former intensity as the inflammatory manifestations disappear.

A valuable therapeutic remedy is vibratory massage and rarefied air applied from the external meatus (Politzer). The instruments used for this purpose are Breitung's or Delstanche's massage apparatus. The latter is particularly suitable for self-application, enabling the patient to apply rarefied air to the external auditory duct at any time, relieving or arresting for the time being the most troublesome symptoms (subjective noises, sensation of fulness in the ear and head, numbness). Massage should not be applied where it is not tolerated, causing momentary exacerbation of the complaints.

Politzer points out that the treatment should not be continued too long and not be instituted more than two or three times a week, as otherwise the originally favorable results may be neutralized. The result after three weeks' treatment may be regarded as the best obtainable.

The subjective complaints are also influenced in some cases by vibratory massage of the nasal and faucial mucosæ, the external auditory duct, and concha; also by faradization of the auricular region.

Should pneumomassage prove insufficient to mobilize the chain of the auricular ossicles, Lucae's pressure sound is to be recommended. The hollow end of the sound is filled with paraffin and applied to the short process after vigorous aspiration, pressure massage being continued for ½–1 minute. Rough movements and excessive pressure should be rigidly avoided.

Strictures due to swelling of the tube can be temporarily relieved by injection of adrenalin solution (adren. 1.0, aq. dest., glycerini āā 1.5).

Extreme retraction of the tympanic membrane which defies any other treatment can be relieved by resecting the posterior fold of the tympanic membrane with a narrow lancet needle, the cutting edge turned upward. This operation should be followed by energetic physical treatment (vibratory massage, aspiration). This will serve to improve permanently the position of the tympanic membrane and consequently the hearing acuity. The same result is sometimes attained by resection of the musculus tensor tympani (tenotomy of the tensor tendon).

V. SIMPLE (SEROUS) ACUTE INFLAMMATION OF THE MIDDLE EAR (OTITIS MEDIA ACUTA SIMPLEX)

Anatomy and Etiology.—In simple acute inflammation of the middle ear there are hyperæmia and œdema of the mucosa, followed in a short time by secretion of a serous or sero-hemorrhagic exudate of the middle-ear spaces. Injection and swelling of the mucosa decrease in a few days, with subsequent complete cure, without any intervening ulceration of the inflammatory tissue or exudate.

Simple as well as purulent otitis media is an infectious disease caused by micro-organisms, which, however, are of slight virility or quite degenerated. Staphylococcus pyogenes aureus, the various forms of streptococcus, and the influenza bacillus predominate. Cases are not rare in which the carefully prepared exudate shows microscopic bacteria, while cultures prove negative.

Mechanical, thermic, and chemical irritation may likewise give rise to simple otitis media. Introduction of irritative vapors into the middle ear through the tube, trauma extending to the tympanic membrane without destroying it, may produce the picture of a serous, though slight, otitis media within a very short time, sometimes within a few minutes.

Infection through the tube may also take place from sneezing, violently blowing the nose, retching or vomiting, syringing the nose, or aspirating water.

In rupture of the tympanic membrane, acute otitis media may occur from entrance of a foreign body into the middle ear, causing traumatic injury to the mucosa. The exudate developing in these cases may be sterile, but soon becomes infected through the tube by the ever-present micro-organisms in the nasopharyngeal tract or through the external auditory duct. Persons suffering from chronic inflammatory changes of the nasopharyngeal tract, especially children with adenoid vegetations, are liable to frequent recurrence of middle-ear inflammation. The various attacks by no means resemble each other, catarrh and inflammation alternating in the clinical picture (Plate VIII, Fig. 6), and the affection may change from simple to perforating otitis media.

Symptoms and Course.—Simple as well as purulent otitis media set in with more or less sudden pain in the ear, impaired hearing, and fever. The pains, which may be severe in the first stage, rapidly subside. Continued severe pain, causing sleepless nights, points to purulent as against simple inflammation. The impairment of hearing is of a medium degree (13½–27 feet C.). Considerable elevation of temperature, which recedes in a few days, is often observed in children.

There are several degrees and forms of swelling and injection of the tympanic cavity, as revealed by the otoscopic examination. Thus, the tympanic membrane may be fleshy and thick, completely hiding the detailed structures including the line of the manubrium, with diffuse hyperæmia of the entire membrane. In other cases, which by no means take a lighter course, the tympanic membrane is only slightly swollen or merely osmotic, dull, and without lustre. In these cases there is radial vascular injection, the red branchlets of the vesicles distinctly showing against the gray background of the tympanic membrane. The circular injection of the peripheral margin is of pathognomonic value.

In influenza and typhoid otitis the epidermal layer of the tympanic membrane and the epithelial covering of the osseous auditory duct are often raised by vesicular formations, the vesicles having usually a bluish-black appearance and containing either blood-serum or a limpid yellow serum (Fig. 69, 1 and 2). Sometimes there are minute extravasations of blood, which cause the tympanic membrane, and in rare cases also the osseous auditory duct, to look as if sprinkled with red dots.

The inflammatory changes of the tympanic membrane are either uniformly distributed over the membrane (mesotympanic type) or they preponderate in the upper part of the membrane, the posterosuperior quadrant, with the membrana flaccida, or the latter alone. In that case we have to deal with the epitympanic type of acute otitis media.

The functional test shows a medium obstacle to sound-conduction without involvement of the labyrinth, provided the hearing function has previously been normal.

Course.—The entire illness usually lasts from 8 to 10 days. The climax is reached in a few days, sometimes as early as the first day. Commencing with the third or fourth day, there are distinct signs of abatement of the inflammatory process, with temperature reduced to normal, the hearing acuity increases spontaneously, and the pains occur only periodically, especially in the evening and after performance of work which causes congestion to the head. In the following few days the tympanic membrane becomes paler, the pains entirely disappear, while the sensitiveness of the tympanic membrane persists for a few days longer. The patient feels the presence of his tympanic membrane, as if the affected ear had not yet returned to normal. Slight catarrhal changes may persist for some time after the inflammation has disap-

Fig. 69.

Formation of vesicles and sacs (prolapse) in serous (1, 2) and purulent (3–5) inflammation of the middle ear. 1. Vesiculation (three vesicles) in the posterosuperior quadrant. 2. Kidney-shaped vesicle of the tympanic membrane. 3. Sac-like dilatation (prolapse) of the posterosuperior portion of the tympanic membrane. 4. Otitis media suppurativa acuta. Considerable swelling and bulging of the entire tympanic membrane. Mammiform protrusion of the membrana flaccida with perforation of the apex. 5. Otitis media suppurativa acuta. Verrucous bulging of the posterior part of the tympanic membrane with perforation.

peared, and the normal lustre of the tympanic membrane and normal hearing acuity may not return until three or four weeks later.

The tympanic vesicles are either evacuated into the auditory canal by spontaneous perforation, or they are resorbed. Hemorrhages, exudates, or blood are inspissated into dark-brown coagula which gradually advance peripherally into the auditory canal.

When the inflammation has run its course, the tympanic membrane may resume its normal condition; in some cases, however, especially in those which run a dragging course, striated, cloudy, or diffuse turbidity of the tympanic membrane, or lime deposits, will develop. The lustre of the membrane is thereby impaired and permanently lost. In these cases the fact should never be lost sight of that the visible changes of the membrane are accompanied by analogous changes in the mucosal folds of the middle ear, which may interfere with the mobility of the chain of auricular ossicles and tympanic membrane, giving rise to permanent, though slight, disturbance of the hearing faculty.

Treatment of the nasopharyngeal tract is absolutely necessary if

the inflammation, which has meanwhile run its course, was caused by chronic changes of that tract. This is the only possibility to prevent recurrence of the inflammatory manifestations, which may resemble the first attack, but often run a much graver course, with ulceration and cerebral complications.

Diagnosis.—Based on the triple symptom-complex—pains in the ear, affected hearing ability, and fever—and assisted by the otoscopic findings, the diagnosis of acute otitis media can be made without difficulty at any stage of the disease.

There are two affections to be considered in the differential diagnosis,—acute inflammation of the tympanic membrane and purulent otitis media. In the former (myringitis acuta) the membrane may exhibit the same changes as in otitis media acuta simplex, but the stabbing pains are only moderate, sometimes there is only an organic sensation of the presence of the membrane, the temperature is not elevated, and the hearing acuity is almost normal. With the aid of the functional findings it is usually quite easy to distinguish the two affections from each other.

The differentiation between simple and purulent otitis media is more difficult. The symptoms in both affections, notably in the first stage, are alike in quality and differ only in degree. Severe pains in the ear which rob the patient of sleep and rest point to purulent as against simple inflammation of the middle ear, even though the otoscopic inflammatory manifestations are not pronounced and the temperature is only moderately elevated. Similarly, considerable impairment of the hearing faculty (3½–7 feet C.) occurs almost exclusively in purulent inflammation of the middle ear, provided hearing has been normal before the last attack. Furthermore, high accessions of temperature favor the assumption of purulent otitis media.

It will thus be easy in many cases to differentiate simple from purulent inflammation of the middle ear at an early stage. Other cases, however, will require one or two days' observation to clear the diagnosis and to determine whether paracentesis should or should not be done. Sometimes, however, it may be impossible even for the experienced specialist to differentiate correctly. This refers especially to infantile cases, where the hearing acuity cannot be exactly determined, and even the objective pains can only be established to a limited extent. The triple symptom-complex in these cases is reduced to the one sign of fever. Considerable elevation of temperature is an important guide for the diagnosis of acute purulent inflammation of the middle ear, and yet there are cases in which the symptom is absent in spite of the purulent character of the inflammation; and in these cases, especially in nurslings, it is the long persistence of the inflammatory manifestations of the

tympanic membrane which points to the purulent nature of the process. The infantile tympanic membrane is thicker than that of the adult, in consequence of which the characteristic purulent inflammatory manifestations (circumscribed yellow discoloration of the membrane and considerable protrusions) develop much later and more slowly than in the adult.

Repeated recurrence of middle-ear catarrh in older children, resulting from chronic changes of the nasopharynx, sometimes cause thickening of the tympanic membrane which partly or entirely prevent the mucous membrane from bulging out. Doubtful cases in children should, therefore, be otoscopically examined several times daily, attention being particularly directed to variations in color and circumscribed red and yellow discolorations.

Treatment.—Insertion of cotton plugs or gauze strips saturated with anodyne or astringent remedies, or corresponding instillations into the affected ear, comprise the treatment in the first stage of simple acute inflammation of the middle ear. The following are such remedies:

Acid. carbol., 0.3–0.5;
Glycer.,
Aq. dest., āā 5.0.

or

Aq. carbol. (1 : 100.0), 10.0;
Cocaini hydrochlor., 2.0;
Atrop. sulph., 0.05. (Lermoyez.)

or

Olei hyoscyami, 0.8;
Olei olivarum, 10.0.

Also, anæsthesin in 2–5 per cent. oleic solution, or alypin 1.0, glycer., aq. dest. āā 15.0. Insertion of cotton tips saturated with warm adrenalin solution has a temporary beneficial effect.

Acetic alumina is particularly suitable for insertion, preferably in the shape of gauze strips saturated with Burrow's solution 1.0, aq. dest. 5.0, or ½ to 15 alsol solution. These substances are less suitable for instillations, owing to their macerating properties.

Application of heat (compresses of acetic alumina 1 : 10, linseed bags, thermophore) has a favorable effect. In other cases, again, cold compresses (Leiter's refrigerator) exert a better effect. Physical and mental rest and copious evacuations are of importance. Aspirin (0.15–0.5), hydropyrin (0.15–0.5), acetopyrin (0.15–0.5), and pyramidon (0.1–0.2) are internal remedies which may have an anodyne effect. These remedies, especially the first two, taken in hot lemonade or hot infusion of tilia flowers, are recommended at the beginning of the disease to produce powerful diaphoresis. Soporifics are of no use; opiates should be avoided, if possible.

The patient should remain in bed as long as the temperature is elevated. As soon as this returns to normal, which is usually the case on the third or fourth day, the involution of the process and resorption of the middle-ear exudate are assisted by the application of Politzer's air-douche. The hearing acuity is tested both before and after air insufflation, and if the acuity is found to have improved, it is an exceedingly favorable prognostic sign.

Opening of the tympanic membrane by paracentesis is indicated if conservative measures bring no relief, the pains continue, and the temperature does not recede to normal. The time for action is determined by the following considerations: Adoption of a waiting attitude without any definite purpose in view is not correct. The fact should always be kept in mind that in doubtful cases it is always better to carry out paracentesis than to neglect it. This minor operation, if carried out under such aseptic precautions as are possible in the ear, is not fraught with any danger. The course of simple otitis media may even be shortened by allowing the serous or sero-hemorrhagic exudate to escape though the aperture made by paracentesis so as to render its resorption superfluous. It is only when paracentesis is done under unclean conditions that the danger exists of transforming a simple into an infectious purulent middle-ear inflammation from infection. On the other hand, omission of timely paracentesis may cause irremediable damage which may lead to severe complications and even death.

For technical instructions to be observed in paracentesis see p. 156.

After the inflammatory manifestations have abated and the tympanic membrane is no longer hyperæmic, treatment of the nasopharyngeal tract must be instituted. Special attention should be given to the faucial tonsils in children. Should they be enlarged and the choanous passages interfered with, the tonsils should be removed as soon as possible.

VI. ACUTE PURULENT INFLAMMATION (ULCERATION) OF THE MIDDLE EAR (OTITIS MEDIA SUPPURATIVA (PERFORATIVA) ACUTA); TYMPANITIS PURULENTA ACUTA

Acute suppuration of the middle ear is very common in children and is caused by bacterial infection. The entrance of the infection is not always clearly demonstrable. Pyogenic factors may possibly find their way into the tympanic cavity through the lymph-channels without infecting the latter, but in most cases the infection occurs through the auditory tube from the nasopharyngeal tract, and the same etiological factors are at work which are responsible for simple inflammation of the middle ear The external auditory duct is only responsible for conveying infection to the middle ear in traumatic suppuration. Acute suppuration of the middle ear in children is frequently observed in the

course of many acute affections of the nasopharyngeal tract and general infectious diseases. The list is headed by suppuration occurring in the course of acute infectious diseases, principally scarlet fever, measles, and typhoid, which are dreaded on account of their grave sequelæ and complications.

Anatomy.—In all cases in which infection of the tympanic cavity occurs through the tube there will be swelling of the tubal mucosa which soon renders the tube impassable. The swelling rapidly spreads to the mucosa of the middle ear and tympanic membrane; all the middle-ear spaces (meso- and hypotympanum, attic, antrum, and often the pneumatic cavities of the mastoid process) are filled with an infectious, viscid or hemorrhagic exudate. Ulceration sets in very shortly, often within a few hours, leading to copious exudation into the middle-ear spaces, the pus eventually perforating the tympanic membrane and escaping through the external meatus. Immediately after perforation the secretion is often blood-tinged, especially in influenzal and typhoid otitis, and will become purulent only at a later stage. After the secretion has lasted for about a week, mucus will be mixed with the pus in the normal course of the disease, the secretion becomes stringy, the quantity of pus diminishes from day to day and is gradually replaced by mucus. When the secretion has been arrested, the aperture of the tympanic membrane closes in normal cases, and there will be anatomical and functional restoration to normal. A few weeks after healing, the macerated epidermal layer of the membrane in children will be desquamated, a new, normal one, with a smooth and lustrous epidermal layer, having meanwhile formed underneath.

Symptoms.—The prominent symptom is violent pain in the ear, especially in the initial stage, which sets in suddenly and without warning. The paroxysms of pain often reach the highest limit, robbing the patient of rest and sleep. The onset of the pain is sometimes preceded by a sensation of occlusion and fulness in the ear and unilateral headache.

In typical, genuine suppuration of the middle ear the hearing difficulty attains to a high degree (3½–7 feet C.); the acuity gradually returns toward the end of the affection and is perfectly normal again by the time the suppuration has run its course.

There is usually considerable fever in the beginning of middle-ear suppuration, often reaching 104° F. and more. With the evacuation of pus after perforation, the temperature sinks by lysis in favorable cases. A sudden drop of the temperature to normal or subnormal in the first days of the inflammation is an unfavorable symptom, unless it is accompanied by an abatement of all other pathological manifestations. Such drops occur in early intracranial involvement, and are sometimes observed in patients whose power of resistance has been reduced by grave

infections. Uncomplicated suppuration of the middle ear often sets in with vomiting, chills, and fever.

Course.—The affection is conveniently divided into three stages:

1. The initial stage, which begins with the onset of ear symptoms and terminates in a short time (sometimes after a few hours or at the most in 3 or 4 days) by perforation of the tympanic membrane and escape of pus from the external duct. Both pain and fever usually continue until perforation and evacuation have taken place.

2. The second stage is characterized by suppuration. There is no pain; the temperature is normal or slightly elevated. There is profuse secretion of pus during the first few days, which spontaneously decreases in about a week. Later the pus becomes stringy, and in the last days the secretion consists of almost pure mucus, the secretion lasting 2–3 weeks altogether. A thick and creamy condition of the pus and the admixture of large quantities of mucus increase the danger of the secretion being retained in the tympanic cavity or deep in the auditory duct, where it desiccates, putrefies, or decomposes. The hearing acuity improves as the suppuration decreases.

3. The final stage sets in with the arrest of the secretion and the gradual filling of the perforation, which will take place in 1–2 weeks. Extensive perforations require more time to heal, but as long as the affection has run a normal course there is no danger of the gap becoming permanent.

Finding of the Tympanic Membrane.—The otoscopic findings show a more or less pronounced œdema and hyperæmia, which may be uniformly diffused over the entire tympanic membrane (Plate VIII, Fig. 7). In other cases the membrane is more inflamed and swollen in some places than in others, where hyperæmia, œdema, and protrusion are particularly pronounced. Extensive vesicle formation is not rare (Fig. 69). In middle-ear suppuration of the epitympanic type (Kuemmel) the inflammatory manifestations are limited to the upper part of the tympanic membrane (Pl. VIII, Fig. 5), Shrapnell's membrane, and the posterosuperior quadrant (Fig. 69, 3–5), and not infrequently lead to wart-like eminences of the posterosuperior quadrant.

Fig. 70.

Acute purulent inflammation of the middle ear during influenza. Three hæmorrhagic vesicles cover the tympanic membrane.

The intensely red color of the tympanic membrane, however, is not a reliable indication of the gravity of the middle-ear inflammation, as the membrane may show a deep-red color and considerable swelling in simple inflammation of the middle ear and even in myringitis. The line of the manubrium is entirely hidden by the swelling. The tympanic membrane may even assume a deep-red coloration in light degrees of

otitis media in the course of influenza, and the epidermal layer may be raised by blisters. On the other hand, the membrane may be pale red even in the presence of considerable accumulation of pus in the middle ear, or there may be only a radial and peripheral vascular injection. Generally speaking, the intensity of the reddening and swelling of the tympanic membrane decreases but little with the progressive purulent transformation of the exudate. If no perforation takes place and the acute inflammation leads to empyema of the middle-ear spaces, especially to empyema of the tympanic cavity, the membrane may be opaque, grayish-yellow, with but a slight injection of the radial vessels.

Imminent perforation is usually indicated by circumscribed yellow discoloration and considerable bulging of the membrane. After perforation there is either a freely passable, visible, more or less circular gap or a fissure, the swollen margins of which lie close together, so that the aperture is only just large enough for the pus to escape. Removing the secretion with swabs may render the perforation invisible, and it is only recognized by the further escape of pus; it may, however, also be localized by aspiration with Siegle's speculum. The aperture is sometimes located at the apex of an eminence.

The size of the perforation varies. Comparatively large apertures with extensive destruction may cause fulminating suppuration of the middle ear in the course of acute infectious diseases, especially scarlet fever.

The findings of the membrane and the mucosa of the tympanic cavity in the advanced stages of suppuration resemble each other in regard to reddening and swelling as well as osmosis. Finally the swelling of the membrane is abated and the epidermal layer dries up. The perforation is now distinctly visible, even in cases where such was impossible before. The margin of the perforation is at first still thicker than the surrounding membrane, but is finally deflated. During the healing process it is colored grayish-white.

The functional test of the auditory duct in all uncomplicated cases shows considerable disturbance of sound-conduction, and the hearing acuity is considerably reduced at the beginning of the inflammation. Great pressure of the secretion is evidently able at this time to cause slight spontaneous nystagmus (bilateral with lateral vision); it disappears as perforation occurs.

Diagnosis.—Based upon the history and the characteristic symptoms (severe sudden pain in the ear, impairment of hearing, findings of the tympanic membrane), the diagnosis of purulent otitis media can be made without difficulty in the first stages of the affection.

For the differential diagnosis there is only simple middle-ear inflammation to be considered. Great swelling of the tympanic membrane, bulging of the posterosuperior quadrant, and especially the circumscribed

yellow discoloration, with considerable impairment of hearing (provided there has been normal acuity previously), point to suppuration as against simple inflammation of the middle ear. The same refers to persistent pain with periodical severe exacerbations. In paroxysmal pain alternating with perfectly painless intervals, however, simple middle-ear inflammation is more probable. High fever points to purulent inflammation of the middle ear. In nurslings and children up to eight years of age it is, of course, impossible to test accurately the hearing acuity; but testing with alarm instruments or well-known noises, such as striking a gong or glass, rattling with keys, high-pitched tuning-forks, will usually establish an approximate determination. In certain cases thorough examination of the antrum (transillumination, X-ray examination) will render valuable service.

Considerable pain which patients are apt to refer to the ear occurs in young children in late teething, acute inflammation of the pharyngeal lymph-glands, tonsillitis, and swelling of the faucial tonsils. Sometimes intense earache occurs as a prodromal symptom of an acute infectious disease, usually following an affection of the nasopharyngeal mucosa. In the period of puberty, violent earache resembles the signs of a beginning otosclerosis, although the otoscopic findings are negative.

In the suppurative stage the odorless, profuse, pulsating secretion, and later the odorless pus mixed with mucus, point to middle-ear suppuration as against otitis externa. In the simultaneous presence of abscesses of the auditory duct (otitis externa furunculosa) it may be difficult to decide at the first examination whether there is simply an inflammation of the auditory duct or a middle-ear suppuration besides, especially if there is a multiple furunculosis of the external duct with considerable secretion of pus.

The following points hold good in regard to differential diagnosis:

1. Otitis externa runs an afebrile course both in older children and adults, while in young children there is sometimes moderate elevation of temperature. Thus, if it is found from the history in doubtful cases that the affection has set in with high fever, there is a probability of the simultaneous presence of middle-ear inflammation.

2. After cleansing the auditory duct in otitis externa and rendering it permeable by the insertion of a thin ear-speculum, there will be normal or slightly reduced hearing distance, provided the tympanic membrane is not too much impaired from a macerated epidermis. In the simultaneous presence of middle-ear suppuration, however, the hearing acuity will not improve even after rendering the auditory duct permeable.

3. The osseous part of the auditory canal is an important guide in children over four years of age. Otitis externa is always confined to the membranous part of the canal, so that the osseous part will be found of

normal width and the tympanic membrane unchanged on inserting the ear-speculum through the stenosed part of the canal. If, on the other hand, there is middle-ear inflammation, and stenosis of the canal due to involvement of the antrum and mastoid process, inspection of the osseous part will reveal a settling of the posterosuperior wall and more pronounced swelling of the membranous than the osseous portion.

Treatment of Acute Suppuration of the Middle Ear.—In the first stage of the affection the treatment should be local and purely symptomatic.

Severe earache is sometimes relieved by instillation of 3–5 per cent. carbolglycerin, solutions of anæsthesin and adrenalin. Hot solutions (104° F.) of acetic alumina or 1 per cent. alsol may be instilled in spacious ducts in which the resultant epithelial maceration will probably cause no constriction. Instillation of cocaine, novocaine, or alypin solutions may have a very good, though only temporary, effect. A cotton tip saturated with a 20 per cent. solution is inserted up to the tympanic membrane. Local application of cold (ice-bag, Leiter's apparatus) is likewise indicated in the first stages of the inflammation. Withdrawal of blood is usually without effect. Rest in bed and copious defecation are essential. Alcoholic beverages and strong tea or coffee are to be avoided.

In considerable pain and fever all anodyne remedies will soon lose their effect. In these cases it is advisable not to wait for spontaneous perforation, but to perform paracentesis without delay. This simple operation is done with the lancet-needle fixed in Politzer's handle. The tympanic membrane is incised from below upward to the umbo. If circumscribed yellow coloration of the membrane indicates imminent perforation, paracentesis should be made at that place, or at least including it, as otherwise there is danger of an unsuccessful operation.

Preparation for Paracentesis.—The concha is cleansed with a cotton tip saturated with benzine, the head is covered with a fenestrated sterile compress, the concha being pulled out through the aperture. The external meatus is cleansed with 6 per cent. peroxide of hydrogen. Instillation of a novocaine-adrenalin solution is to be recommended for inducing anæsthesia, 1 c.c. of a 20 per cent. novocaine or alypin solution, heated to 104° F., being added shortly before use to 5 drops of the heated adrenalin solution (commercial). The fluid is allowed to remain in the external duct for 10–15 minutes. The Freiburg Ear Clinic recommends the injection of a Schleich-adrenalin mixture into the tympanic cavity.

Instillation of a 5–10 per cent. aqueous cocaine solution, with an addition of 5 drops adrenalin solution, 1.0 : 1000, has also been recommended. Eucaine (8 per cent. aqueous solution) is non-toxic and sterilizable. The following should also be mentioned: Gray's solution

(cocaine muriat. 0.5, ol. anil., alcohol absol. āā 5.0 : 10 drops, heated to 104° F., are allowed to remain in the auditory duct for about 5 minutes); Haug's solution (cocain. muriat. 1.5–3.0, aq. dest., glycer. āā 10.0, alcohol absol. 10.0).

The operation should be made under aseptic precautions to avoid the danger of secondary infection. If paracentesis is done at the proper time, it will be followed immediately by a sero-hemorrhagic or a hemorrhagico-purulent exudate. Immediate evacuation of pure pus shows that the operation was done too late, that the inflammatory exudate has been completely transformed into pus, and that all middle-ear spaces are replete with pus (acute empyema of the middle ear). Gauze strips, prepared with hot acetic alumina or 1 per cent. alsol, should be held in readiness for immediate insertion into the auditory meatus after paracentesis, without resorting to drying or syringing. A moist ear compress follows, and the patient is put to bed. In favorable cases there is profuse purulent secretion from the external meatus in the course of the next few hours. The temperature gradually recedes to normal in from 3 to 5 days; the pain usually disappears completely a few hours after paracentesis.

If the perforation is not located on the level membrane, but at the apex of an eminence, there may be purulent stagnation leading to temporary agglutination and retention. In these cases a liberal incision of the eminence with the lancet-needle is the indicated treatment.

Commencing purulent secretion marks the second stage of the affection, and the only thing to do now is to keep up the evacuation of pus and prevent its retention or stagnation. This is accomplished in two different ways. First, moist compresses of acetic alumina or 2 per cent. alsol are applied and renewed in 24 hours. A moist gauze compress is introduced into the external meatus for purposes of drainage, and after 24 hours all the layers of the compress are usually saturated with pus. Later hydrophile or antiseptically impregnated gauze strips, 4–6 cm. long and 2 cm. wide, are inserted (xeroform, iodoform, dermatol, aristol, ectogen). A nurse can attend to the renewal of the gauze strips, using straight or geniculate forceps with blunt points, which are cleansed with benzine and sterilized by boiling both before and after use. The strips have to be renewed at regular intervals: in extensive secretions every one or two hours, in slight secretions two or three times a day. The strips should not be allowed to remain in the ear when no longer able to absorb secretion; as soon as a strip is saturated, it has to be removed before the secretion behind has had a chance to stagnate. A sign of sufficiently frequent renewal of the strips is the presence of very little pus in the external meatus upon removal of the strips, and the fact that the patient is free from pain and experiences a free sensation in the

external duct. Irrigations are not required in perfectly normal cases; but when the secretion is dry and tough, sterilized water of a temperature of 100°–104° F. should be used. Insufflation of antiseptic powders is quite superfluous, especially where the secretion is copious.

This method of treatment also has the advantage of keeping the physician informed on the condition of the suppuration, although he may not see the patient daily, the decrease in the amount of secretion being shown by the gauze strips becoming less saturated and finally remaining dry. In the normal course, the purulent secretion becomes more viscous at the end of the first week, and a few days later stringy, since the mucoid pus has a tendency of depositing itself in the tympanic cavity or external duct. At this period application of peroxide of hydrogen is indicated (3–6 per cent. solution of hydrogen peroxide, or perhydrol 5.0 : aq. dest. 30.0), which is instilled into the ear 3 or 4 times daily at a temperature of 104° F. The pus is now removed from the tympanic cavity 3 or 4 times a week by air insufflation, followed by cleansing of the external duct with sterile cotton tips and 5 per cent. aqueous perhydrol solution. The hearing acuity usually rapidly increases under the influence of air insufflation, and undergoes a temporary reduction only when the middle-ear spaces are again replete with secretion. Aspiration of the secretion by careful rarefaction of air in the external duct with Siegle's speculum or an aspirating bulb may be serviceable. Great care and attention should be devoted to the external ear, since an additional eczema or inflammation of the auditory duct will favor the pus stagnation in the middle ear. In the last stages of the secretion, insufflation of air should be resorted to daily until the hearing acuity remains permanently on a satisfactory level and the lateral surface of the tympanic membrane has lost its hyperæmic appearance and become dry. The gauze inserts are continued until the perforation is completely closed. After the arrest of the secretion, the perforation gap usually closes within a few days, the first sign being white coloration of the margin by the young, newly growing epidermis.

Fig. 71.

1. Intermediary (free) lime deposits in the tympanic membrane. 2. Central (fixating) lime deposits in the tympanic membrane. 3. Peripheral (fixating) lime deposits in the tympanic membrane.

While the gap closes, it becomes gradually smaller. The new growth of tissue occurs almost exclusively from the central part of the tympanic membrane to the periphery. Local treatment in this stage is to be deprecated. The perforation closes quite spontaneously, and the tympanic membrane as well as the hearing acuity becomes normal again.

Middle-ear inflammation which has persisted for a long time may

be followed by scars of lime deposits in the tympanic membrane (Fig. 71). After the middle-ear suppuration has run its course, it is advisable to keep the external meatus closed for several weeks by a small cotton tampon.

In order to prevent recurrence of middle-ear inflammation or catarrh, it is necessary to treat the nasopharyngeal tract and to institute therapeutic measures to ensure permeability of the choanæ and nasopharynx as well as to care for restitution of normal conditions of the pharyngeal openings.

VII. ACUTE INFLAMMATION OF THE MIDDLE EAR IN INFANTS AND YOUNG CHILDREN (ACUTE INFANTILE OTITIS)

Otitis media of the nursing period has latterly received close attention by Gomperz. Valuable contributions on this frequent affection have also been contributed by Aschoff, Politzer, Preysing, and S. Weiss.

The occurrence of otitis media in infancy is principally favored by the anatomical conditions of the infantile auditory duct. The great frequency of infantile otitis is explained, according to Gomperz, by the following etiological factors: (1) The tubal ostia open into the nasopharyngeal space; (2) the changes in the circulation following birth, together with hyperæmia and loosening of the tympanic mucosa; (3) persistence of the embryonal character of the middle-ear mucosa beyond the fetal period; (4) immaturity; (5) hereditary constitutional affections, such as scrofulosis, tuberculosis, syphilis, rhachitis, and descent from alcoholic parents; (6) traumatic injury of the middle ear before and during birth, entrance of amnion or its constituents into the middle ear; (7) infectious diseases, especially la grippe, less frequently measles, pertussis, varicella, diphtheria, scarlet fever, in the course of which coughing, sneezing, crying, and vomiting lead to infection of the middle ear; (8) hæmatogenous infection; (9) gastro-intestinal and cerebral affections, which lead to infection of the tympanic cavity from vomiting.

The tympanic cavity of infants contains abundant mucous tissue and plenty of fluid in the first few weeks of life. Both are excellent culture grounds for invading bacteria. The myxomatous tissue is subject to very rapid purulent transformation, and, as there are vast accumulations of it in the upper tympanic cavity, epitympanic suppurations in infants and young children are of frequent occurrence and by no means devoid of danger. During the same period the middle-ear spaces are not freely permeable. The epitympanum and the upper part of the mesotympanum are infiltrated with connective-tissue bridges and mucosal septa; the folds of the mucosa are extremely thick and succulent; the antrum contains plenty of mucous tissue in the first weeks of life; and thus it comes to pass that with the onset of an infection a large

number of disseminated small inflammatory foci will develop in the tympanic cavity. These inflammatory foci partly intercommunicate and are partly isolated and segegated by the mucosal septa. Under these circumstances there is very little chance for early and convenient escape of the pus, once purulent transformation has begun. It is only after fluidification of the entire mucous tissue of the middle ear and after formation of a true empyema of the tympanic cavity that spontaneous drainage can be hoped for. Up to that time extensive swelling and deep ulcerations of the middle-ear mucosa will develop. The occurrence of characteristic papillary proliferations of the inflamed mucosa of the middle ear has been established by Politzer.

The Eustachian tube of the new-born is remarkably short and wide, and the muscles are feebly developed. Nor can it be definitely denied that in some cases the Eustachian tube is not completely closed at birth. It is through all these conditions that infection of the infantile middle ear on the part of the pharynx is particularly favored. It may easily happen that the mucus present in the pharynx, aspirated fluid, etc., may enter the tympanic cavity of infants by deglutition and vomiting. In many cases otitis may occur from the fact that the penetrated mucus is at the same time the carrier of the infection; in other cases the mucus entering into the middle-ear spaces may act as a foreign body, causing as such an inflammation which is changed into ulceration by secondary infection from the pharynx; indeed, many authors look upon infantile otitis as an affection due to the entrance of foreign bodies.

Suppuration is further favored by the fact that debilitated infants, who are particularly liable to infection, suffer from impaired respiration and deglutition, and that in tender and anæmic children accumulation of mucus or food remnants in the pharynx cannot be avoided.

One of the principal causes of the frequent occurrence of middle-ear inflammation in infants and young children is the frequency of faucial and tubal affections. Furthermore, the recumbent position of nurslings is to be taken into account, as it favors irritation of the tube and consequently the middle ear in disturbed deglutition or respiration, and at the same time fluids, coagulated milk particles, etc., may enter the tympanic cavity. Furthermore, there seems to be a possibility that in infants and young children up to the age of two or three years, who suffer from a serious affection, there occurs spontaneous suppuration of the mucous tissue in the middle ear owing to general debility and anæmia. In these cases the micro-organisms which are always present in the nasopharyngeal tract, but cannot bring about a purulent inflammation in a healthy child, may lead to suppurative decomposition of the mucous tissue of the tympanic cavity, which is an easy prey to infection and purulent transformation, as explained above. This also

explains why, in autopsies on infants who died during the first month of life up to the end of the first year, suppurative exudates are often found in the tympanic cavity, and why the histological examination of these cases admits of distinct demonstration of acute inflammatory changes of the mucous membrane. Abundant inflammatory foci are especially found in the mucous tissue accumulated at the fundus of the tympanic cavity and in the attic.

The healthy tympanic cavity is germ-free, according to the investigations of Preysing and S. Weiss. Infantile otitis is caused in the majority of cases by the pneumococcus.

Symptomic Peculiarities in Infantile Otitis.—The most important point is that in infants and young children there may exist not only catarrhal affections, but also purulent inflammation of the middle ear, with apparently light (or without any) symptoms, in spite of serious conditions. Besides there is the danger that fever, the most constant prodromal symptom of acute inflammatory affections of the nasopharyngeal and respiratory tracts, is not referred to the ear, but erroneously to affections of the tracts mentioned. Gomperz rightly recommends examining the ears of infants and young children in all febrile affections, even in the absence of ear symptoms.

The possibility of overlooking an inflammation or even suppuration of the middle ear is increased by the helplessness of the infant. It is not before the fourth month that infants direct attention to the possibility of an auricular affection by rubbing the ear, putting the hand to the head, crying whenever the ear or its vicinity is touched, and even avoiding to lie down on the affected side. Exacerbation of the pain in the ear during sucking often interrupts the feed, the infant giving vent to crying. Up to that age, however, all motor reaction may be absent, and attention is aroused only by the occurrence of purulent secretion. This, however, occurs at a comparatively late stage, the tympanic membrane of the new-born being thicker and more resistant than that of the adult. Sometimes it takes as long as one or two weeks for the tissue of the tympanic membrane to become ulcerative, causing perforation and evacuation of the pus through the external meatus. Furthermore, the late occurrence of perforation is favored by the fact that the permeable tympanic cavity at that age does not represent a uniform space, so that time is required for a sufficiently large quantity of pus to accumulate which is capable of powerfully bulging out the tympanic membrane, stretching the tympanic tissue, and thus accelerating the purulent transformation and perforation. The latter may also be delayed or frustrated by the pus escaping into the pharynx through the short and ample tube.

I observed the case of a girl, 12 years of age, with a chronic sup-

puration which had existed for years. The tympanic membrane had never been perforated and revealed nothing but two intermediary lime deposits; evacuation of the pus occurred spontaneously and without trouble into the pharynx through the Eustachian tube.

Suppuration of the middle ear occurs less frequently in breast-fed than bottle-fed infants. There is sometimes danger of otitis in breast-fed infants from unsuccessful sucking if both nares are occluded by pressure from the mother's breast and milk particles enter the tympanic cavity in swallowing.

Owing to congenital cracks of the osseous facial canal, there is greater danger in infantile otitis than in otitis media of the adult, of peripheral paralysis of the facial nerve, due to spreading of the inflammation to the connective tissue enveloping the nerve; but any such paralysis is only slight and will disappear in a few days or, at the most, two or three weeks. Faradic excitability is nearly always preserved, and there is no degenerative reaction.

I am unable to share the opinion that otitis runs a more fulminating course in robust than in under-nourished and debilitated children, and that the perforation in the former occurs rapidly and in the latter slowly or not at all. The fact that at autopsy pus has been frequently found in the middle ear of infants, but rarely a perforation, may be explained in this way, that otitis in these cases developed during agony, so that there was no time for perforation to take place. Besides, in a number of these cases the suppuration of the middle ear seems to be of a tuberculous nature, and delayed perforation under these circumstances also occurs in adults.

An extremely characteristic symptom of acute infantile otitis is the sudden onset of fever, in which the temperature reaches the highest possible degrees in the first few days. Temperatures up to 104° and 106° F. are by no means rare. The fever is of the continuous type, and return to normal or subnormal temperature is usually a sign of complications.

In the first days of serious inflammation of the middle ear, chills and fever, sometimes accompanied by vomiting, will occur just before perforation. Apparently meningitic symptoms may likewise set in, such as collapse manifestations, stupor, lagophthalmos, nasal respiration, sudden unrest, epileptoid movements of the extremities, crying out, disturbed sleep, or sleeplessness. Ortner has summarized this symptom-complex under the name of "meningism." These grave manifestations sometimes disappear after spontaneous perforation or paracentesis, as soon as the pus commences to escape. Should they occur while purulent secretion is already established, it is the first sign of positive meningitis with unfavorable prognosis.

Peculiarities of the Course of the Affection.—A few important points may be mentioned. Purulent secretion may occur later in infants and young children than in adults, but may also last much longer. This does not signify a disturbed course, nor give rise to apprehension, unless there are objective signs of an involvement of the mastoid process. This is in many cases due to the fact that inflammation of the middle ear preferably attacks debilitated, under-nourished, rhachitic, or otherwise affected children, especially those who have been weakened from acute infections and recovered but slowly.

A particularly unfavorable symptom of infantile otitis is abundant granulation of the middle ear and of the perforation margin of the tympanic cavity, which may occur at an early stage. This condition may interfere with free evacuation of the pus from the middle ear, causing it to be retained in the middle-ear spaces; sometimes the secretion is completely arrested. Such retention is always associated with recurrence or exacerbation of the local pains and elevation of temperature, together with a further decrease of the hearing acuity.

A further peculiarity of infantile otitis consists in the great danger of abscess formation in the pars mastoidea, which is favored by the relatively large antrum being but loosely connected with the tympanic cavity, and by the plethoric diploic bone of the pars mastoidea in infants and young children. These abscesses very rapidly perforate outward, forming a subperiosteal mastoid abscess, the lateral wall of the antrum being a very thin osseous layer which often contains cartilaginous remnants in rhachitic children. The cartilage rapidly ulcerates, a fistula resulting at the lateral surface of the pars mastoidea and underneath the periosteum.

Finally, it is not surprising that tuberculous suppuration of the middle ear, which is not rare in infancy and childhood, sets in without symptoms and may remain undiscovered for a considerable time. This condition prevails to a certain extent even in adults, and is only accidentally noticed when pus exudes from the ear or impaired hearing makes itself felt.

The resorptive properties of the infantile mucosa of the middle ear are unquestionably greater than those of the adult, and comparatively extensive exudates may still be resorbed in the various stages of infantile otitis.

Tabes mesenterica which is sometimes associated with otitis media has been divided by Preysing into two groups: (1) that attributable to the respiratory tract and (2) that attributable to the intestinal tract. He does not believe, however, that this affection results from aspiration or escape of the secretion from the middle ear into the respiratory or intestinal tract, but rather that it is due to toxic substances which have found their way into the blood and lymph currents owing to rapid resorption from the middle-ear empyema.

Otoscopic Examination and Findings.—Formerly many authors considered otoscopic examination of the infantile tympanic membrane to be impossible, and we are indebted to Gomperz for having demonstrated the possibility of methodic otoscopy of infants. He devised a set of 5 particularly short ear-specula of ½, 1, 2, 3, and 4 mm. in diameter, respectively, for irrigation of the infantile ear, the cannula being covered by a short draining tube.

Cleansing the external auditory duct from scales or cerumen has to be done most carefully, the infant's head being held perfectly quiet. After insertion of the speculum, the otoscopic examination can be facilitated by pressing it downward toward the base of the auditory duct. It should be remembered that the infantile duct consists only of the membranous part, that it becomes narrower as it approaches the tympanic membrane, and only widens in a kind of fissure immediately before the membrane. The latter inclines considerably outward, so that its posterosuperior part is closer to the eye of the examiner than the anterior section to an even greater degree than is the case in adults. Gomperz also states that the infantile tympanic membrane assumes a pink tint while the infant is in the act of crying.

Blood extravasations of the tympanic membrane are no less frequent than in adults. In considerable œdema of the epidermal layer the tympanic membrane has at times a pale-red (grayish-red) tint, even in the presence of considerable inflammation. Occasionally several eminences are visible. The line of the manubrium is entirely absent. Blood vesicles can often be observed in the initial stages of influenza otitis. After perforation has set in, especially in the posterosuperior part, wart-like or conical protuberances are by no means rare. The margins of the perforation are usually considerably swollen, with the result that the perforation can be recognized as a gap on outflow of pus or aspiration. Occasionally granulations develop at the margin of perforation in the course of a few days, with polypoid protrusions toward the external duct. As is the case in the adult, profuse suppuration is followed later by a mucous secretion. With the arrest of the secretion the gap usually closes within a few days, while the granulations at the margin of perforation undergo spontaneous involution in proportion to the quantitative diminution of pus. Operative removal of the granulations is only necessary when the healing process takes a dragging course.

The difference in the otoscopic findings between otitis with acute infections, and tuberculous otitis media, has been discussed in the chapter on the Diseases of the Ear and Acute Infectious Diseases.

Diagnosis, Prognosis, and Course.—With painstaking observation of the patient and careful otoscopic examination, there can be no dif-

ficulty in the diagnosis of infantile otitis, provided the pædiatrist considers the possibilities of otitis media in infants and young children at a sufficiently early stage. The prognosis should be made cautiously. Relatively slight hyperæmia of the tympanic membrane in infants by no means excludes the purulent character of otitis media. After complete development of an empyema of the middle ear, the fever may abate or entirely disappear without perforation of the membrane, but spontaneous perforation may still occur later. It is advisable, therefore, to postpone the decision of the prognostic question, as to whether a perforation is to be expected or not, until either the tympanic findings definitely indicate the imminent perforation (circumscribed red or yellow coloration) or the involution of the inflammatory manifestations is recognizable by a rapid reduction of the swollen membrane.

The prognosis is favorable in otherwise robust, well-nourished infants for both simple and suppurative otitis media. The organ will be completely restored without any impairment of the hearing acuity. In genuine inflammation of the middle ear and in otitis developing in the course of common colds (coryza, bronchitis, etc.), a permanent aperture need not be apprehended.

In debilitated, anæmic, under-nourished, rhachitic infants there is some danger of acute otitis media developing into the chronic form, and in the most favorable contingency into healing at a later period, but with permanent changes remaining. This is particularly to be apprehended if the initial stage of suppuration has escaped attention or has been neglected. When the suppuration has become chronic, it soon leads to moist eczema and constriction of the auditory duct, with consequent retention of pus in the middle ear, ulcerous processes of the mucosa, formation of granulations and polypi, carious changes of the auricular vessels and osseous walls of the middle ear, with all their sequelæ.

The prognosis is unfavorable in tuberculous infantile otitis and ulcerations of the middle ear caused by the streptococcus mucosus. Endocranial involvement is rare in infantile age, probably owing to the fact that, after involvement of the mastoid, outward perforation underneath the periosteum occurs more rapidly than in older children or adults.

The treatment of infantile otitis hardly differs from that of otitis media in adults. Early paracentesis is important, as its omission may be responsible for the sudden development of a grave cerebral symptom-complex (meningism), or suppurative meningitis. The auditory duct requires the greatest care; the skin should be repeatedly anointed and the lumen kept free by thorough removal of the secretion and the macerated epithelial masses which accumulate in the form of small scales. Constriction or occlusion of the auditory duct by eczema, inflammatory

processes, etc., may considerably hinder the treatment and cause retention of the secretion, with incalculable consequences. In a protracted course repeated paracentesis is advisable. Extensive granulations may be removed at an early period; should they be allowed to remain, the inflammation will usually take a longer course and may even become chronic from proliferation of granulations. After the inflammation of the middle ear has been cured, it is often necessary to remove the considerably enlarged palatal and faucial tonsils to prevent recurrence of the inflammation.

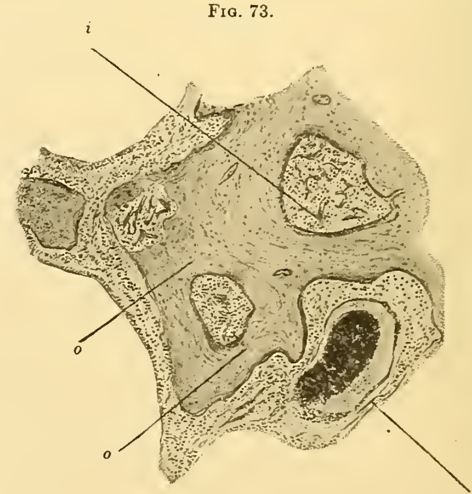
VIII. ACUTE PURULENT MASTOIDITIS (OSTEOPERIOSTITIS OF THE MASTOID PROCESS)

Anatomy.—The anatomical changes in acute mastoiditis depend upon the kind of infection, the duration of the illness, and the normal anatomical conditions of the mastoid process. The beginning of the inflammation is marked by hyperæmia of the mastoid mucosa and the inflammatory thickening of the soft covering of the mastoid process (lateral wall of the mastoid and posterosuperior wall of the osseous auditory duct). The blood-vessels are considerably distended. Ulcerative transformation of the inflammatory exudate and abscess formation occurs as the inflammation proceeds. This condition leads to empyema in a pneumatic mastoid (Fig. 72). The various cells become replete with pus, which soon extends over all mastoid cavities corresponding to the normal intercommunication of the mastoid cells. Purulent decomposition of the osseous septa and for-

Fig. 72.

Acute mastoiditis with suppuration in a mastoid air-space. Boy twelve years old.

Fig. 73.



Acute mastoiditis (diploic mastoid). Boy six years old. *o*, normal bone; *i*, inflammatory infiltrate; *a*, pus.

mation of a confluent abscess filling the entire mastoid process will develop later, usually in a few weeks. In diploic mastoids small disseminated abscesses, from millet-seed to hemp-seed size (Fig. 73), will develop first, the diploic tissue soon becomes ulcerative, and the final result is again a large abscess. As early as the end of the first week granulations will commence to develop in the abscess cavity or mastoid cells, as the case may be, which will steadily increase; the walls of old abscesses are sometimes completely covered with granulations.

Fig. 74.

Typical cleft of the external auditory duct and descending posterosuperior wall of the duct (*a*) in acute purulent mastoiditis.

Occasionally there is spontaneous healing of the abscess by resorption or escape of the pus into the tympanic cavity through the antrum and thence outward through the perforation aperture. The extension of the suppurative inflammation of the mastoid to the squama and the zygomatic process is discussed on page 281.

Perforation of the abscess through the walls of the mastoid process is, however, of far greater frequency. If it occurs through the lateral wall, a localized subperiosteal abscess will develop in the mastoid region (Figs. 76, 77); if through the anterior wall, purulent infiltration of the integument of the auditory canal with eventual perforation into the canal will be the result.

Fig. 75.

Descending posterosuperior wall of the auditory duct (*a*) and cleft-like constriction (*b*) of the duct in acute suppuration of the antrum and pars mastoidea in a child three months old.

In tuberculous abscesses it is not a rare occurrence for the auditory duct to be surrounded by pus, and for the abscess eventually to perforate through the anterior wall of the duct at the fusion of the os tympanum with the membranous part of the duct or through the gap of ossification of the os tympanum toward the sub-maxillary articulation. The final result in these cases may be purulent inflammation of the articulation.

Downward perforation of the abscess will cause descending abscesses to develop, the various forms of which may be summarized under the generic name of Bezold's mastoiditis.

As the pus finally advances through the medial wall of the mastoid, it will lead to endocranial involvement (pachymeningitis externa, extradural abscess, etc.).

In the early stages of mastoiditis the interior of the mastoid is considerably more affected than the periosteum; acute tuberculous mastoiditis, however, usually runs its course under the picture of osteo-

periostitis, the first symptom principally involving the periosteum with early granulation of the periosteal part confronting the bone.

The consistency of the pus contained in the mastoid depends upon the infection and time of the primary illness (middle-ear suppuration). In streptococcus and staphylococcus infections the pus is yellow and fluid; in tuberculous infections it is exceedingly thin or even watery, greenish-yellow, and infiltrated with thick pus flakes. Streptococcus mucosus produces a mucous pus of slight fluidity, and the influenza bacillus leads to hemorrhagic abscesses. Fetid abscess contents point to grave infections (scarlet fever, measles, diphtheria) or to chronic general affections (tuberculosis). Fetid pus is also found in mastoiditis occurring in

Fig. 76.

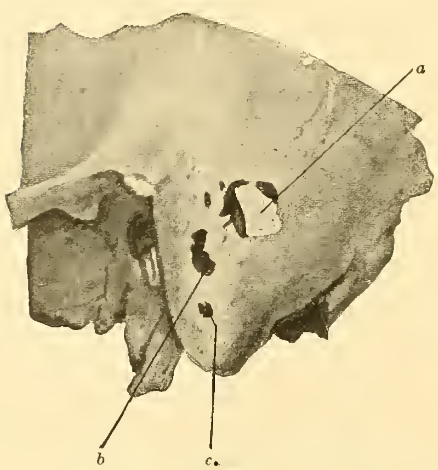
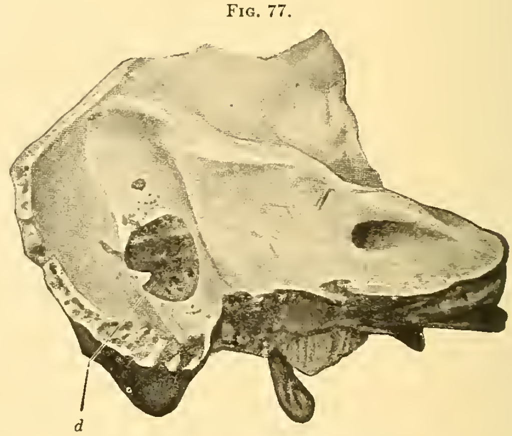


Fig. 77.



Purulent mastoiditis with multiple fistula formation (*a*, *b*, *c*) and fistulous perforation into the sulcus sigmoideus (*d*).

the course of chronic suppuration of the middle ear. Gaseous abscesses of the mastoid process are rare, being found only in cases where the abscess has resulted from chronic suppuration of the middle ear.

With a mastoid abscess enclosed on all sides, the pus is under considerable pressure, and immediately exudes upon resection, sometimes under pulsation. This would indicate that the mastoid abscess has advanced to the dura or that an extradural abscess has already developed. Where the abscess communicates with the antrum there can be no question of any particular pressure within the abscess.

Etiology.—There are primary and secondary purulent mastoiditis to be differentiated. Primary mastoiditis is very rare. It occurs from the entrance of pyogenic factors into the mastoid itself, where they cause inflammation, while the other middle-ear spaces either remain uninvolved or merely show catarrhal changes of the mucosa. Such a localized effect of the infectious germ is favored by preceding traumatic injury to the

mastoid. There are greater chances for it to occur where there has been a previous inflammation than where the mastoid has always been healthy.

Typical secondary mastoiditis is of much more frequent occurrence, and is due to the spreading of a middle-ear suppuration which had so far been confined to the tympanic cavity and antrum, to the mastoid process. The involvement of the mastoid occurs either after the type of continuous extension (chiefly in the mastoid air spaces) or after the type of metastatic suppuration (chiefly in the diploic mastoid). In the former case the mastoid abscess is in communication with the rest of the middle-ear spaces from the beginning; in metastatic abscess formation the abscess may at first be closed on all sides, and a communication with the other middle-ear spaces is only created by perforation of the abscess into the antrum. As a matter of course, there are many transition forms, which is intelligible from the fact that the mastoid is only rarely pneumatic or diploic throughout, and that in the majority of cases there is a so-called mixed mastoid, containing diploic as well as pneumatic regions.

Mastoiditis and middle-ear suppuration occur at widely different periods. Anatomical examination of cases of acute otitis media where the suppuration has existed only a few days shows, to the surprise of the examiner, that the mastoid cells nearly always contain pus. It is evident that in a large number of cases pus enters the mastoid from the antrum in certain positions of the head or body, or through other physical causes, without giving rise to any material co-inflammation of the mastoid. In most cases these changes will rapidly return to normal, so that at the end of the first week of the middle-ear suppuration the mastoid region is again normal and free from pain.

The typical acute purulent mastoiditis sets in during the three or four weeks of acute middle-ear suppuration after more or less prodromal signs, at a time when the secretion from the external auditory duct usually still persists.

Occasionally the symptoms of mastoiditis will not occur until the close of the perforation and arrested secretion from the external duct, but the impairment of hearing acuity will indicate that the tympanic cavity is by no means normal yet.

Acute mastoiditis occurring in the course of chronic suppuration of the middle ear is exclusively due to direct spreading of the pus to the mastoid process. The ultimate cause is usually acute suppuration of a cholesteatoma, or acute retention owing to stenosis of the auditory duct or polyp formation. In many of these cases the endocranium will be involved, either simultaneously or at a later period.

Symptoms.—According to localization there are to be distinguished: (1) symptoms of the mastoid itself, (2) other ear symptoms, (3) cerebral symptoms, (4) general symptoms.

1. *Mastoid Symptoms.*—In the first place there is swelling of the soft covers of the mastoid. Thickening of the lateral periosteum can be established by comparison with the normal side. It can be distinctly felt that on the affected side there is between the palpating finger and the osseous surface a thicker soft layer than on the healthy side. The physician takes up a position behind the patient and palpates bilaterally with the first and second fingers between mastoid and concha, trying to advance in the direction of the crista temporalis inferior. In advanced cases the skin over the mastoid is tense, glistening, and hyperæmic. There is also spontaneous and pressure pain of the mastoid, especially at the apex. The inflammatory (collateral) œdema of the soft mastoid covers leads to cleft-like constriction of the external auditory duct. The anterior wall of the mastoid and antrum forms at the same time the posterosuperior wall of the osseous external duct. Inflammation of the interior of the mastoid causes swelling of the periosteal investment of the auditory duct at this spot and descent of the posterosuperior osseous wall of the auditory duct (Fig. 75). At the same time the anterosuperior wall of the membranous part is passively stretched, so that the transverse section of the duct has the form of a fissure running in an anterosuperior to a postero-inferior direction (Fig. 74). The characteristic point is that in advanced cases the swelling increases toward the tympanic membrane and that the latter cannot be inspected owing to the cleft-like constriction of the auditory duct, the deep part of the latter being impassable for the speculum.

The œdematous swelling of the soft mastoid covers leads to the pathognomonic change of position of the concha. The concha is deflected laterally and is at a lower level than on the healthy side, and there is antero-inferior torsion (Figs. 78, 79). This position anomaly is particularly striking in the posterior aspect (Fig. 79).

2. *The Other Ear Symptoms.*—Special signs may be absent. Reduced hearing distance (3½–7 feet C.), which persists for weeks, points to considerable accumulation of secretion and a further dissemination of the inflammatory changes in the middle-ear spaces. In other cases a suspicious sign, setting in shortly after the secretion has begun, consists in the great quantitative variation of the evacuated secretion. On some days or at certain hours it is above normal, while at other times it is very slight or absent for a shorter or longer period, although the other inflammatory manifestations (fever, pain, impaired hearing) continue.

In a case of acute suppuration of the middle ear which runs a normal course, the pus secretion presents a characteristic picture: the tympanic membrane having been perforated, the secretion rapidly increases during the first twenty-four hours, remains at the same level during the next few days, and, aside from variations which are not accompanied

by pain or fever, decreases in quantity with spontaneous improvement of the hearing acuity. As the quantity of the secretion decreases, the pus is more and more infiltrated with mucus, and as the inflammatory manifestations abate, it becomes stringy and inspissated.

The acute diminution of the secretion is often observed at just the time that the local mastoid signs intensify. In the presence of pus retained in the tympanic cavity, there are violent, throbbing ear pains, like those occurring in the beginning of otitis media.

In other cases again profuse otorrhœa, which persists for weeks, and obstinate earache point to the involvement of the mastoid process.

Fig. 78.

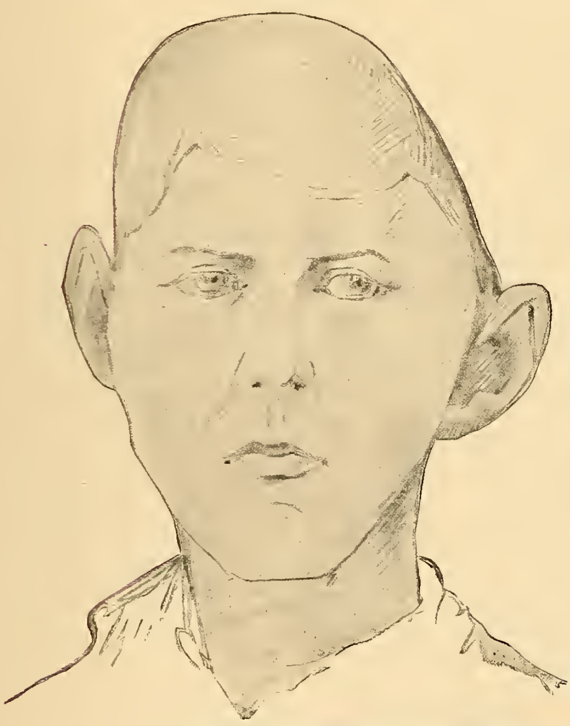


Fig. 79.

Pathognomonic position of the concha in acute purulent mastoiditis. Boy 14 years old.

3. *The cerebral symptoms* consist in diffuse headache, which is usually localized toward the affected side. Sometimes there are vomiting, vertigo, or convulsions in the initial stage.

4. Among *the general symptoms* elevation of temperature should be mentioned first. It is principally present in the first stage of mastoiditis, later in circumvallate mastoid abscesses. Febrile ascents up to 104° F. and more are observed in children in the beginning of mastoiditis. Variations of temperature are frequent, and depend upon the free or impeded escape of the secretion. Fever may be entirely absent if the

abscess is perforated or communicates from the first with the antrum and the tympanic cavity. Immediate fall of the temperature to normal or subnormal, followed by renewed access of fever, cannot be explained by simple mastoiditis, but rather points to intracranial involvement and, above all, to an affection of the venous sinuses. The frequency of pulse and respiration is increased in proportion to the fever. The general impression of the patient chiefly depends upon his subjective complaints. If there are considerable regional pain, general physical unrest, especially at night, lassitude, anorexia, bad complexion, the patient gives the impression of being a very sick man. On the other hand, cases in which there are no subjective pathological manifestations whatever are by no means rare, even in the presence of a large mastoid abscess.

Unusual and general symptoms give rise to the suspicion that mastoiditis is not the only affection caused by the underlying infection. In the presence of chills, delirium, icterus, localized headache, impaired motility of the head and vertebral column, we shall not be content with the diagnosis of "inflammation of the mastoid process," but it is our duty to search for another accompanying affection,—above all, an endocranial involvement.

Diagnosis.—Taking all the above symptoms into consideration, there can be no difficulty in arriving at a diagnosis of acute mastoiditis. Inspection of the patient from the base of the skull (Fig. 79) is of importance. The abnormal position of the ear will then be at once apparent in cases in which it may escape the eye of the observer in the anterior aspect. The examination has to be repeated, if necessary. It stands to reason that the symptoms are not alike in degree and intensity in all cases, and it may require very careful examination and considerable experience to guard against overlooking an inflammation. If the mastoid manifestations are slight, suppuration from the middle ear lasting for more than four weeks and continuously affecting the hearing ability points to an abscess of the mastoid process. In the absence of any other manifestations, the very descent of the posterosuperior wall of the osseous duct may lead to the correct diagnosis.

In doubtful cases transillumination of the mastoid may render good service. Two small cold electric lights of special construction are introduced into the two auditory ducts. The normal mastoid allows a fair quantity of light to shine through, the pneumatic mastoid appearing light red and the dipolic mastoid deep red. The posterior border of the diaphanous part usually coincides with the hair border in the normal individual, while in inflammatory affections of the mastoid the pathological side is much less permeable to light than the healthy one, and the shadow border will be a considerable distance in front of the hair border. Unimportant deviations in the diaphanic properties, however, do not constitute any point of diagnostic value.

The diagnosis of acute mastoiditis may also be aided by X-ray examination, although any mastoid changes thereby disclosed would be of no diagnostic value unless supported by clinical symptoms.

The difference in temperature between the affected and the healthy mastoid regions is of diagnostic value, although important and easily recognizable differences will only be present after the other local symptoms are fully developed. It requires considerable experience to recognize slight differences.

Differential Diagnosis.—There are three affections to be differentiated: (1) otitis externa furunculosa; (2) erysipelas of the auricular region; (3) pediculosis capitis with lymphangitis of the scalp, as well as moist eczema and furunculosis of the scalp.

Simple furunculosis of the auditory duct may cause far-reaching œdema of the auricular region, resulting in pasty swelling of the covering of the soft mastoid parts, perhaps hyperæmia of the skin, and in rare cases in fluctuation, the latter occurring particularly in deep ramified abscesses and tuberculous inflammation of the external ear.

In these cases the differential diagnosis presents no difficulties. In the first place, there is either slight or no painfulness, spontaneous or on pressure. Since furuncles of this kind are usually situated at the base of the external meatus, the base of the concha is raised by the œdema, causing the entire concha to assume a higher position than normal (Figs. 63*a* and 63*b*). Nor is the œdema confined to the mastoid region, spreading as it does to the parotid region and in typical cases to the lower eyelid. Upon examination of the affected external duct no changes will be found in that region except those of positive furunculosis (circumscribed swelling, painful pressure point at the tragus).

The fissure-like constriction in acute mastoiditis is particularly conspicuous in the region of the osseous duct, while in inflammation of the external auditory duct the constriction is confined to the membranous part of the duct. If in otitis externa a small ear-speculum has passed the constriction, it is very easy to establish with the aid of the otoscope the fact that the osseous duct is normal and that the tympanic membrane and the hearing acuity have undergone no change, provided the external duct is maintained in a permeable condition by means of the funnel. On the other hand, the patient will experience pain in mastication, which is not the case in true mastoiditis (except in inflammatory ankylosis due to perforation of the abscess toward the maxillary articulation). The temperature is either normal or but slightly elevated in otitis externa.

The differential diagnosis becomes more complex if there is also otitis media in addition to otitis externa, and inspection of the tympanic membrane is prevented by granulations or accumulated crusts, or if

the hearing acuity is reduced owing to maceration of the epidermal layer of the tympanic membrane. In these cases, however, the decision will be rendered possible either by repeated careful examination and observation for one or two days, or in cases of ulceration by the operative findings.

Cranial erysipelas, spreading to the auricular and mastoid regions, may give rise to manifestations which resemble those of acute mastoiditis. The history (not a preceding affection of the ear), together with the normal auricular findings, will protect against error, while a precise examination at daylight will disclose the characteristic demarcation of the dermatic swelling.

Pediculosis capitis, notably of the vertex and occiput, may be accompanied by inflammation of the lymph-vessels, with swelling of the skin and painful swelling of the mastoid glands. Examination of the auditory duct, the exact history, and, above all, the bilateral involvement of the mastoid region in pediculosis capitis will render a correct diagnosis possible.

Treatment.—Conservative treatment of acute mastoiditis is chiefly supported by appropriate treatment of the middle-ear inflammation, preventing stagnation of the secretion and avoiding unnecessary irritation. Should signs develop pointing to involvement of the mastoid process, absolute rest in bed is advisable. Cold compresses (ice-bag, Leiter's apparatus) usually render good service in the beginning of the affection, but at later stages patients usually prefer warm applications (thermophore, cataplasms). If compresses prepared with acetic alumina or 1–2 per cent. alsol, or alcohol, are used, the ice-bag or thermophore is applied laterally over the compress, with patient in the dorsal decubitus. Painting with iodine or applying silver ointment (ung. colloidale) has furnished no convincing proof of usefulness. Bier's hyperæmia deserves a trial with patients who can be constantly observed in a hospital. I have never seen any very remarkable results from this proceeding, but it appears that in appropriate cases involution of the inflammatory mastoid manifestations is favorably influenced. But a curative effect cannot be expected from Bier's hyperæmia in cases where suppurative transformation of the tissue and abscess formation in the mastoid have already taken place. On the contrary, cases which had been subjected to this procedure and which later had to be operated upon usually gave the impression as if hyperæmic stasis were responsible for the rapid spreading of the pus to all the cavities of the mastoid and even for the perforation of the abscess.

Resection of the mastoid is indicated if there are no distinct signs of an abatement of the mastoid symptoms after all conservative measures have been exhausted.

Indications.—The indications are perfectly clear in cases which have been observed from the beginning of the middle-ear inflammation and where the development of mastoiditis could be plainly followed. Operative interference is a matter of necessity where, in spite of appropriate treatment, the development of mastoiditis could not be prevented.

The decision is more difficult in cases of acute mastoiditis which could only be observed after prolonged existence of otitis media which has not had the benefit of competent treatment or of any treatment at all. In the absence of severe symptoms, such as great pain, high temperature, etc., a waiting attitude under strict supervision may be justified for a time. Every experienced specialist is aware of the fact that cases of this kind, presenting the manifestations of mastoiditis, may undergo involution with rest in bed and local treatment. There may be nothing but inflammatory irritation which disappears as soon as the patient is given the opportunity of rest and care, such involution proceeding more rapidly in some cases than in others.

Cases of entirely circumvallate mastoid abscesses may suddenly come to a favorable termination, as I have repeatedly had occasion to observe. Thus, a girl 14 years old, suffering from suppurative middle-ear inflammation, developed manifestations of acute mastoiditis in the third week. Conservative treatment gave no improvement, but, at the urgent request of the parents and in the absence of threatening symptoms, this was continued for three weeks. As there was no improvement, pains and swelling persisting, operation was finally decided upon for the following morning. During the night the abscess perforated into the antrum and thence outward through the opening of the tympanic membrane. Eight days later the patient was completely restored.

The value of the separate symptoms of mastoiditis, so far as indications for operation are concerned, stands in relation to the duration of the middle-ear suppuration. It is not a rare occurrence that mastoid symptoms (pain on pressure, slight swelling of the soft covers) occur in the beginning of otitis media as partial manifestations of a suppurative inflammation in the tympanic cavity and antrum, which would indicate that the mastoid manifestations represent a distinct effect of the middle-ear inflammation. As a rule, these symptoms disappear within a few days after the pus has been properly evacuated by paracentesis.

In the course of acute infantile infections, mastoid symptoms in the beginning of acute attic suppuration or in otitis media are of especial importance, as unsuccessful paracentesis and continuation of the mastoid manifestations would indicate involvement of the mastoid process. As a rule, the symptoms are not fully developed until the fourth week.

In prolonged cases, on the other hand, the symptoms to go by are often very few. Thus, important local mastoid signs may be absent

as soon as the abscess has perforated toward the dura. In these cases the indication for operation is continued poor hearing and persistent dropping of the posterosuperior wall of the auditory duct. In cases, however, where the middle-ear inflammation has been cured, the indication for operation is given by persistent pain in the mastoid region, with headache, and continuous though slight elevation of temperature. The importance of bacteriological findings is not to be underrated, but it need not be emphasized that the microscopically and culturally demonstrable micro-organisms must emanate from a secretion taken under sterile precautions from the tympanic cavity in the beginning of the inflammation (on the occasion of paracentesis). On the other hand, no reliable result can be expected from the pathological examination of pus spontaneously exuding through the external meatus. Should the presence of streptococcus mucosus, S. pyogenes, or bacillus pyocyaneus be demonstrated, early operation is advisable.

The age of the patient likewise is a point to be considered. Up to 8 years the mastoid abscess is nearly always in communication with the antrum. The spontaneous evacuation into the tympanic cavity in infancy is, therefore, a far more likely occurrence than in older children or adults. Besides, the lateral antrum wall is thin, in rhachitic children often cartilaginous in parts, so that a spontaneous perforation of the abscess outwardly under formation of a subperiosteal abscess is to be expected early, and the danger of the mastoid abscess perforating toward the dura under a waiting attitude is comparatively slight.

In older, anæmic children who have been weakened by other illness, the formation of extensive mastoid abscesses often presents but slight local symptoms. The periosteal swelling of the lateral wall may be slight or entirely absent in the presence of a thick corticalis.

Grave constitutional or advanced organic affections contraindicate resection of the mastoid in uncomplicated suppurative mastoiditis. In cases of this description operation is desisted from (1) on account of dangerous postoperative possibilities and (2) owing to the probability of unfavorable healing, accompanied, as it usually is, by profuse secretion, leading to rapid marasmus. In diabetes there is besides the danger of coma diabeticum immediately following operation.

In advanced tuberculosis, in general debility, or in bilateral involvement in bottle-fed infants, operation should only be resorted to if there is impending danger to life by the affection spreading from the mastoid to the interior of the cranial cavity.

Method of Operation.—(1) *Simple Resection of the Mastoid* (*Mastoidotomy*).—The planum mastoideum is exposed and the corticalis removed with the chisel and Luer's bone-forceps, corresponding to the lateral surface of the mastoid process. Chisel work commences

in the mastoid triangle (Fig. 80). The abscess having been evacuated and the softened osseous parts removed, the edges of the corticalis are resected far enough to let the traumatic cavity in the bone form an obtuse angle with the corticalis. Should the mastoid apex seem to be involved, it is advisable to detach the outer part of the tendon of the sternocleidomastoid and to remove the apex with Luer's bone-forceps. An iodoform, isoform, or simply sterile wick is inserted into the wound. In this way the traumatic cavity is loosely packed and the skin closed

FIG. 80.

FIG. 81.

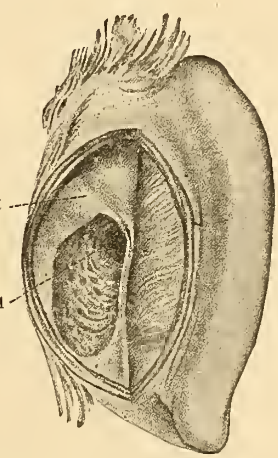


FIG. 80.—The mastoid triangle (*a*, blue) results from the linear connection of the torus supramastoideus (*Tsm*), the tuberculum suprameatum (*Tsme*), and the mastoid apex. The antrum triangle (*b*, red) results from the linear connection of the torus supramastoideus and the tuberculum suprameatum with the lowest point of the porus acusticus externus. ($\frac{2}{3}$ natural size.) *Cti*, crista temporalis inferior.

FIG. 81.—Right ear. *A* antrum mastoideum; *Cti*, crista temporalis inferior. ($\frac{2}{3}$ natural size.)

by sutures or Michel's clamps up to the drainage angle, carefully avoiding the periosteum. A sterile gauze strip is inserted into the previously cleansed auditory canal.

(2) *Opening the Antrum; Antrotomy.*—The operation is commenced in the same way as simple resection of the mastoid. The bone is chiselled off in an anterosuperior direction, closely following the direction of the upper auditory wall (Fig. 81). Small pneumatic cells indicate the immediate proximity of the antrum, as does the escape of air bubbles. Should the abscess cavity extend to the middle wall of the mastoid, it is advisable to resect the bone edges and the external margin of the osseous posterior wall of the auditory duct.

The wound is attended to in the same way as after mastoidotomy. Scraping the resected antrum may easily lead to luxation of the incus and should therefore be avoided.

Should the healing process take a regular course without reactions, the first change of bandages should take place on the sixth day, the

wicks being shortened at the same time. During the second week the bandages are changed daily, with gradual shortening of the wicks, so that with the third or fourth change all the wicks are removed. Gauze strips are now inserted into the auditory meatus and tympanic cavity. After-treatment of the resected antrum need not be troubled about; repeated sounding, cauterization, or irrigation will interfere with the healing process and may give rise to a permanent antrum fistula. As a rule, healing takes place by the traumatic cavity filling up with granulation tissue, which finally changes into connective tissue. In young, vigorous individuals there will be new-formation of the cortex from the periosteum, if preserved. Regeneration of the osseous trabecula within the mastoid itself occurs but very rarely. It is inadvisable to attempt obliteration of the mastoid connective tissue and new-formation of the cortex in debilitated individuals or where the traumatic cavity is unusually extensive or deep. The skin should rather be pulled into the traumatic cavity by simple traction sutures, with a view to effecting direct union of the epidermal layer with the osseous wall by tamponade under moderate pressure, although the cosmetic effect will be impaired by a deep scar in the mastoid region. But this is compensated for by the solid healing, while in primary suture down to the lower traumatic angle the conditions referred to—debilitation and deep traumatic cavity—may easily cause the healing to take place at the level of the skin, with a chronic abscess remaining below, which may perforate many months later and require scraping or renewed operation. It is for this very reason that "healing under the blood scab," as advocated by some authors, with complete primary closure of the skin wound, is objectionable, destroying as it does the possibility of controlling the healing process, and possibly leading in apparently healed cases to very unpleasant surprises, suppuration of the blood scabs weeks afterward, and formation of new abscesses.

The average time required for healing after simple resection of the mastoid process is 3–4 weeks, after antrotomy 5–7 weeks. Should the healing process take a dragging course, it may be stimulated by illumination of the traumatic cavity (sunlight, Auer lamp, cold electric bulbs introduced into the cavity) and scarlet red ointment. Silver nitrate ointment is not recommended, as silver nitrate even considerably diluted will almost without exception cause suppurative relapses if any part of it reaches the tympanic cavity through the open antrum. However, when the traumatic cavity is entirely obliterated and the antrum completely closed, the final closure of the skin wound can be accelerated by the nitrate stick. The healing process will also be aided by nutrition, avoidance of physical efforts, and fresh air. Sojourn at the sea-coast and sea-baths often have a remarkably favorable effect.

Uncomplicated cases of mastoiditis in which the middle-ear suppuration has not been of long duration will heal with complete anatomical and physiological restoration of the middle ear: tympanic cavity, membrane, and hearing distance will become normal again. If, however, the middle-ear suppuration should have lasted for several months, there may be cicatrization of the tympanic membrane and development of connective-tissue layers in the tympanic cavity as well as permanent reduction of the hearing acuity.

Simple resection of the mastoid will not answer the purpose unless the bone toward the antrum is absolutely intact. Should the bone be affected, or in doubtful cases, antrotomy should be performed.

Wilde's incision in acute mastoiditis has been rightly abandoned. It may perhaps be in place to relieve any considerable tension of the covers of the soft parts (in subperiosteal abscess) where local conditions will not permit of immediate bone operation, but then it is only a temporary help preparatory to the bone operation.

Acute infantile mastoiditis is characterized by early spontaneous perforation outward and formation of a subperiosteal abscess. The majority of the cases are recruited from under-nourished, weak infants of from patients suffering from acute otitis running along with scarlet fever or measles.

The youngest infant I have observed with purulent mastoiditis and subperiosteal abscess was three months old. In one infant seven months old, bilateral mastoiditis with subperiosteal abscess developed in the course of one week.

Suppurative infantile mastoiditis is quite often caused by tuberculous middle-ear suppuration, the former developing in the shape of tuberculosus osteoperiostitis, whereas purulent osteoperiostitis of the temporal squama and the superior wall of the auditory canal occurs less often. Owing to secondary pneumo- or streptococcus infection, extensive subperiosteal abscesses may here be formed which may finally reach the occiput and base of the skull posteriorly and the lateral canthus of the eye anteriorly.

IX. CHRONIC MIDDLE-EAR SUPPURATION

1. SIMPLE CHRONIC MIDDLE-EAR SUPPURATION

The simple forms of chronic middle-ear suppuration are those in which the pathological process has been confined to the mucosa of the middle ear in a prolonged course of the affection without spreading to the bone.

In chronic suppuration of the mucosa the latter has become swollen and softened, leading to chronic ulceration and granulation, possibly to the development of granulating polyps. On the other hand, the

occurrence of myxomatous polyps enclosed in an epidermal layer, as well as the presence of cholesteatoma, is evidence of the inflammation having spread from the mucous membrane to the osseous parts of the middle ear.

Etiology.—Simple chronic middle-ear suppuration develops from the acute form if circumstances prevail which cause the inflamed, purulent state to continue, preventing the reduction of the inflammation in the acute or subacute stages. These causes may lie in the middle ear itself: perforation in the acute stage of otorrhœa may be prematurely constricted or occluded by granulation; or the acute middle-ear suppuration may have occurred after the pathological septa had been formed in the tympanic cavity from previous catarrhal affections. Free evacuation of pus in the acute stage of purulent inflammation is impossible in either case, the consequence being that the mucosa undergoes extensive ulceration owing to decomposition of retained pus. These ulcerations behave differently according to which part of the middle ear they occupy. Thus, it may happen that the pathological process in one part of the tympanic mucosa may heal while the ulcerations spread to a hitherto healthy portion.

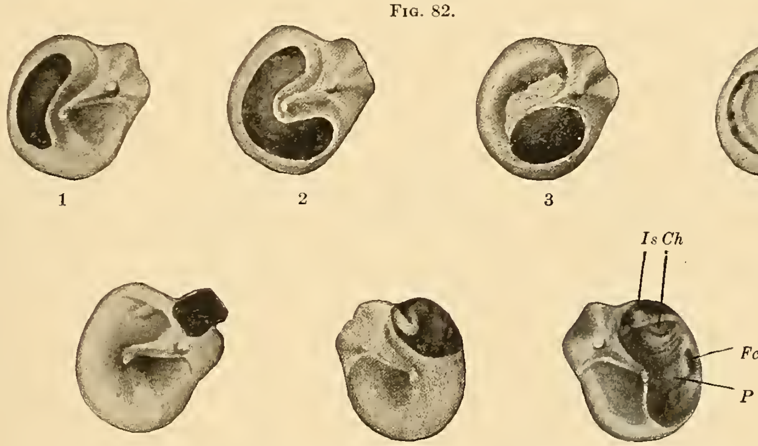
In other cases the chronic suppuration persists owing to catarrhal changes in the nasopharyngeal space. Insignificant causes, such as a simple rhinitis, may be sufficient to develop an acute tubal catarrh on the basis of chronic catarrhal changes, rekindling the old middle-ear suppuration on each occasion. The consequence is that the slight quantity of pus still present becomes profuse (pulsating pus), or suppuration may start afresh after the secretion had been temporarily arrested. The latter kind of relapse is of frequent occurrence in children.

There is also danger of chronic middle-ear suppuration when a weakly, anæmic, or tuberculous patient, with little power of resistance, contracts acute inflammation of the middle ear. In acute infantile cases the danger of chronic development is therefore especially great, as two unfavorable factors coöperate: (1) an acute inflammation running a severe, violent course, with rapid destruction of the soft parts, and (2) impaired power of resistance owing to a previous infectious disease (scarlet fever, measles, etc.).

Tuberculous middle-ear suppuration shows from its very beginning a tendency to a chronic course, and the same danger prevails in the epitympanic forms. This perforation is limited in size by the smallness of Shrapnell's membrane (Fig. 82, 5), and the upper tympanic cavity is difficult to drain, due to the position of the auditory ossicles and the numerous folds of the mucosa. These forms, however, are but rarely confined to the mucosa, and usually spread to the osseous parts of the epitympanum.

Symptoms.—The pathological picture throughout presents the symptoms of chronic otorrhœa. The consistency of the secretion varies: in simultaneous catarrhal changes of the nose and nasopharyngeal space it is often mucous; in the presence of granulations and polypi (Figs. 82, 83, and Plate VIII) it is often hemorrhagic. The pus is usually fetid in neglected cases, never in those properly treated. The condition of the tympanic membrane is at once characterized by the presence of the perforation, which embraces several smaller or larger parts of the membrane and presents the most variegated configurations (Figs. 82, 83, Plate VIII). The manubrium is intact in many cases, and the margin of the perforation does not reach up to the insertion of the tympanic membrane. In other cases the larger part of the membrane and manu-

Fig. 82.



Tympanic membranes in chronic middle-ear suppuration. Right side 1–5; left side 6 and 7. 1. Kidney-shaped perforation. 2. Heart-shaped perforation with preserved manubrium. 3. Oval perforation the size of a hemp-seed; antero-inferior aspect; central lime deposits fused with the manubrium. 4. Large roundish defect of the tympanic cavity; the background of the perforation is furnished by the promontory. 5. Perforation of Shrapnell's membrane; epitympanic chronic suppuration. 6. Marginal perforation of the posterosuperior quadrant. The incus-stapes connection, the upper part of the promontory and of the fenestra cochleæ are visible in the perforation gap. Chronic suppuration of the antrum. 7. Marginal kidney-shaped perforation of the two posterior quadrants. The following parts are visible in the perforation gap: chorda tympani (*Ch*), incus-stapes connection (*Is*), fenestra cochleæ (*Fc*), and the promontory (*P*).

brium is destroyed. Lime deposits of varying extent can often be observed. Again, the lateral surface of the tympanic membrane may be ulcerated (chronic myringitis ulcerosa), or the epidermal layer of the remaining part of the membrane is intact while the remnant itself is of a dull or grayish red. In chronic middle-ear suppuration with temporarily arrested secretion there may be profuse ligament formation in the middle-ear spaces in intermittent periods of healing (Plate VIII). These ligaments are apt to lead to synechiæ of the remnant of the membrane or manubrium with the wall of the tympanic cavity, to obliteration of the corners of the fenestra, or, finally, to obliteration of the tympanic cavity itself (chronic adhesive process following chronic midde-ear suppuration).

The mucous membrane of the tympanic cavity is hyperæmic, swollen, covered with pus, and often ulcerative. Granulations or polypi from hempseed to pea size are likewise encountered in the hypo- and epitympanum (Figs. 82, 83, and Plate VIII). The mastoid region is normal.

The auditory canal is intact in simple chronic middle-ear suppuration; the mucosa of the osseous duct is neither lividly discolored nor ulcerative. The duct itself as well as the mastoid process is unchanged, and the chain of auricular ossicles is preserved. Pus can either be freely evacuated or whatever retention there may be can be easily overcome. There is no cholesteatoma.

The subjective symptoms consist in a feeling of fulness in the ear or head and in subjective noises. There may be pain in the presence of extensive ulceration or pus retention. The funnel test shows all the signs of an obstacle to conduction, with medium or high-grade reduction of the hearing acuity. In degeneration of Corti's organ there are also

FIG. 83.

Tympanic membranes of the right ears in chronic middle-ear suppuration after acute infections. 1–3. Chronic middle-ear suppuration after scarlet fever. 4–5. Chronic middle-ear suppuration after measles. 1. Kidney-shaped perforation of both posterior quadrants. At the base of the perforation the long pillar of the incus, the fenestra cochleæ and the promontory are visible. 2. Heart-shaped perforation with handle of malleus intact. The junction of the stapes and incus, the promontory and the fenestra cochleæ are visible through the perforation. 3. Total destruction of the pars tensa of the tympanic membrane. The short process of the malleus and the membrana flaccida are intact, the incus is destroyed. Through the perforation the head of the stapes and the fenestra are visible. The promontory, tympanic opening of the tube and the ridge of the hypotympanum are to be seen. 4. Complete destruction of the tympanic membrane with absence of incus and stapes. The posterior half of the field of vision is filled with granulation tissue growing from the hypotympanum. The remainder of the field is swollen and congested. 5. Heart-shaped defect in the membrane with malleus and both anterior quadrants intact. The incus is destroyed. The stapes, fenestra cochleæ, chorda tympani and the promontory are visible through the perforation.

the clinical signs of an affected internal ear, consisting in considerable reduction of the hearing acuity, lowering of the upper sound limit, shortened perception of high sounds, and shortened bone-conduction.

Chronic middle-ear suppuration is often accompanied by degeneration of Corti's organ at the base of the cochlea, which, according to anatomical findings, consists in connective-tissue proliferations emanating from the base of the cochlea and leading, as a secondary manifestation, to degeneration of Corti's organ and the cochlear nerve of the vestibular portion. This degenerative atrophy of Corti's organ may gradually spread to the other parts of the cochlea.

Diagnosis of Simple Chronic Middle-ear Suppuration.—The foundation of the diagnosis is supplied by the otoscopic findings (Plate VIII and Figs. 82, 83) and the history. In order to assume the presence of

uncomplicated chronic suppuration of the mucous membrane, as against the surgical forms, the entire auditory canal must be demonstrably intact. Exclusion of cholesteatoma requires microscopical examination of the pus evacuated from the middle-ear spaces, always observing the precautions described on p. 199. Several days or weeks of observation may be required to determine whether the suppuration has been confined to the mucous membrane or whether it has attacked the bone. Pus retention is usually indicated by annoying head pressure, headache, earache, and, possibly, elevation of temperature. There is a purulent secretion in the middle-ear spaces, rapidly accumulating behind the drain, sometimes within a few minutes after the most careful cleansing, which, however, can be aspirated with Siegle's funnel immediately after the examination. A healthy condition of the bone is indicated by the fact that the fetid character of the suppuration completely disappears within a few days under antiseptic treatment energetically directed to drainage and systematic cleansing of the middle-ear spaces.

Treatment.—Conservative treatment is indicated in nearly all cases of simple chronic middle-ear suppuration. Pathological tissue proliferation of the middle ear, granulations, or polypi, if present should first be removed surgically. Otherwise conservative treatment, as outlined below, should be instituted at once. Removal of the polypi and auricular ossicles is merely a preliminary procedure to render conservative treatment effective by making the occluded middle-ear spaces accessible.

(a) *Removal of Granulations and Polypi.*—Local anæsthesia, as described on p. 97, is employed in the removal of polypi, either by cauterization or the knife. If the latter is used, cauterization of the base of the polypus is done one or two days after removal. By far the best cauterizing agent for the middle ear, by reason of its positive effect, is crystallized chromic acid (chromic acid pearl) which has been united with the tip of a probe by melting over a flame. None but flat granulations which are clearly visible in the otoscopic picture are suitable for cauterization. Large granulations or polypi are best removed with small forceps or snares. There are cutting snares and pressure snares, the former cutting through the polypus at the place of application, while constricting loops firmly encircle the polypus and usually detach it at the base when traction is made. It is, therefore, easier with the latter instrument to remove a polypus in one sitting than with the cutting snares; but there is danger of traumatic injury of the base of the polyp from sudden tearing off of the growth. For this reason, constriction snares should not be used for polypi which originate in the superior cavity, as the previously affected osseous roof of the tympanic cavity may be easily injured and develop a suppurative meningitis.

No irrigation of the middle ear should be done immediately after removal of a polypus. The auditory canal and the middle ear are packed as thoroughly as possible with iodoform gauze, and a bandage is applied.

The gauze strips are removed in 24 hours; hemorrhage, if any, is arrested with cotton tips saturated with adrenalin. The stump of the polyp, which must be distinctly visible in the otoscope, is cauterized with chromic acid on the same or the following day.

(b) *The Conservative Treatment of Chronic Middle-ear Suppuration.*—The most important object of the treatment consists in removal of the secretion from the middle-ear spaces and drying up the pathologically changed secreting mucous membrane. The secretion may be removed either by irrigation or dry cotton tips. The secretion collected in the tube is conveyed to the middle ear by Politzer's air insufflation and removed.

The irrigation is effected either with sterilized water or with medicated fluids, such as hydrogen peroxide (1–2 tablespoonfuls of a 6 per cent. solution to ½ litre of water); permanganate of potash, up to a light violet color; lysol or lysoform; formalin (5 drops of a concentrated solution—42 per cent.—to ½ litre of water) argyrol (1 : 1000); alsol (1 teaspoonful to ½–1 litre of water); sublamine (1 : 1000).

The middle ear having been cleansed, antiseptic or astringent medicaments are applied, such as instillations of hydrogen peroxide, perhydrol (Merck) in 3–6 per cent. solution, or, if the mucosa is swollen and granulated, alcohol heated to 104° F. Alcohol is used owing to its hygroscopic properties in withdrawing water from the tissues and causing hyperplastic mucosæ to shrink. As alcohol usually causes a burning sensation, it should be first applied diluted, gradually using stronger solutions. Additions of 1–2 per cent. boric or salicylic acid are also useful. Alcohol, however, should be discontinued if it causes intense hyperæmia or swelling of the affected mucosa.

Introduction of Peru balsam or concentrated aqueous picric acid solutions sometimes exerts an excellent effect. E. Urbantschitsch recommends thigenol (a composition of sodium and sulpho-oleic acid), owing to its anæmic, resorptive, and desiccating effect.

℞. Thigenol "Roche," 5.0;
Spir. vin. dil., 30.0.

or

Thigenol "Roche," 5.0;
Glycerin, 10.0;
Perhydrol, 3.0;
Spir. vin. rectif., 30.0.

The following are astringent solutions:

1. Acid. tannic. 0.5 : 25.0 glycerin, aq. dest. āā.
2. Zinc. sulf. 0.05–0.2 : 20.0 aq. dest.
3. Alum. crud. 0.1–0.2 : 20.0 aq. dest.

In cases of moderate secretion, medicated powders in small quantities are blown upon the mucous membrane. This process should not be left to the patient or his attendants, since exaggerated quantities may cause a dry crust to form with consequent retention of the secretion. In using powders a change should be made from time to time, using alternately acid. boric. subtil. pulver., borodate, xeroform, airol, almatein, and iodol (particularly in tuberculosis). Politzer recommends the following in blennorrhœal secretions: Acid. boric. subtil. pulveris. 5.0, Ol. terebinth. gtt. v.

Very good results are sometimes obtained in obstinate cases with instillations of 3–10 per cent. silver nitrate solutions or insertion of corresponding cotton plugs, after the middle-ear mucosa has been carefully cleansed. Should there be great pain, the ear is immediately rinsed with tepid water. Instillation of a few drops of electrargol or insertion of cotton plugs saturated with this substance is sometimes very effective. Silver treatment is contraindicated in considerable granulation of the mucous membrane, in suppuration of the accessory spaces of the tympanic cavity, and in acute exacerbations.

In favorable cases there is often rapid diminution of the secretion in the course of a few days; cases where the secretion retains its fetid character in spite of careful cleansing and treatment are not suitable for conservative treatment.

The decrease of the secretion can be observed by the patient or attendants by renewing the hydrophile or antiseptic gauze strips at regular intervals.[1] As improvement progresses, there will be no more secretion during the day, while the night strips are partly moistened in the morning; finally this moisture is likewise arrested. However, the dry condition of the strips will not justify the conclusion of the secretion being completely arrested: careful inspection may reveal a small crust or scab, the removal of which will start the secretion afresh. In these cases there is no need for inspecting the middle-ear spaces, as the strips will have a more or less objectionable odor in spite of their being perfectly dry.

As soon as the secretion has definitely subsided, the mucous membrane usually returns to normal in a short time. The formation of epithelium is sometimes assisted by the insertion of a 3 per cent. epicarin or a 1 per cent. scarlet red ointment once or twice weekly.

After the arrest of the secretion nothing but sterile gauze strips should be inserted, which are renewed both in the morning and evening for purposes of control. Cases which lend themselves to conservative treatment are cured in from three to six weeks.

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[1] There are a large variety of antiseptic gauzes, such as xeroform and dermatol, isoform, airol, vioform, aristol, europhen, almatein, ectogen, loratin, and argentol.

(c) *Closing of Persistent Perforations of the Tympanic Membrane.*—In cases of long duration or great virulence of the pathogenic factors, defects of the tympanic membrane of variable extent may persist in spite of careful treatment. Ear specialists have endeavored for a long time to effect a closure of these defects, not only because they impair the hearing acuity, but also because of the risk of a new infection of the middle ear being caused by the exposed condition of the mucosa.

Among the methods used for this purpose are multiple scarifications of the edges of the perforation, removal of the thickened edges by the knife, silver nitrate cautery or galvanocautery; but they are seldom attended with success. Berthold succeeded in closing small and middle-sized perforations by a process of skin transplantation which he called "myringoplastic." The method most frequently applied consists in cauterizing the edges of the aperture with trichloracetic acid. This substance was discovered by Domas in 1839, but it was not until 1889 that v. Stein called attention to its caustic and astringent properties at the Paris Medical Congress of that year. Baratoux (Paris) first used it for closure of dry perforations of the tympanic membrane. In 1895 Oknuff again called attention to this remedy and especially to its value in the scarification of tympanic perforations.

Gomperz and Wassmund recommend cauterization with a concentrated trichloracetic acid resulting from fluidification of the crystallized substance; Urbantschitsch and Biehl recommend 10–50 per cent. solutions. A cotton plug immersed in a 10 per cent. sterile cocaine solution is placed to the remnant of the tympanic membrane for 10 minutes; next the edges of the perforation are touched with a little cotton immersed in the solution and fastened to the tip of a probe. This will cause a whitish corrosive scab, sometimes accompanied by hyperæmia of the tympanic membrane and more or less violent pain. In some cases a mucous or serous secretion follows the operation. Further cauterization is only permitted after all reactions have subsided. Generally speaking, the cauterization should not be done oftener than in intervals of five to eight days. According to the size of the aperture, closure will occur after three to fifteen applications. Should a crust form at the margin of the perforation, it will have to be removed with a pincet or probe before cauterization is repeated.

In case of considerable induration of the margins, it is advisable, according to Miot, to apply galvanocautery or to make multiple radial incisions prior to the application of trichloracetic acid. The same author advises to protect the newly-formed scar from tearing in the first few weeks by inserting a cotton plug and interdicting forced expiration. Heermann recommends scarlet-red salve to effect a reduction of tympanic perforations, this substance leading, according to his observations,

to progressive growth of the tympanic membrane within a few days—so much so that he had to interrupt the treatment in order not to produce exaggerated irritation. In other cases he found it advantageous to extend the irritation to the rest of the tissue elements by additional applications.

All these procedures can be successful only after the pathological process of the middle ear has entirely run its course. Premature cauterization leads almost invariably to rekindling of the inflammatory process. It should further be considered that in many cases the cicatricial closure does not constitute an improvement, but a deterioration of the auditory acuity, which is even liable to cause subjective ear noises. Politzer recommends that, previous to closure of a perforation, small perforations should be tentatively filled up with a little drop of diluted glycerin, larger ones closed with a moist piece of sterile tissue paper, and then the hearing acuity be tested. The result will show the success to be expected. Should the hearing acuity be less than with the open perforation, or should there be ear noises, the aperture should not be closed.

(d) *Endotympanic Measures.*—While a chronic middle-ear suppuration is in the process of healing, scabs or multiple cicatrices may develop in the middle ear. In other cases there may be adhesions (synechiæ) between the manubrium or remnant of the tympanic membrane and the middle wall of the tympanic cavity, especially in the region of the promontory. With an intact or nearly intact labyrinth, endotympanic incision of the cicatrices or removal of the synechiæ in these cases may lead to considerable and permanent improvement of the hearing acuity. Should the windows of the labyrinth be covered with pathologic connective-tissue layers, resection or removal of the latter in the region of the cochlear or vestibular window often has a strikingly favorable effect upon the auditory acuity.

Gomperz divides the adhesions between the edges of the perforation and the wall of the tympanic cavity with a synechotome, and afterward cauterizes the edges with trichloracetic acid, with the result of effecting a considerable improvement in hearing.

(e) *Artificial Tympanic Membranes.*—Should the attempt at closing the perforation be unsuccessful or the membrane be entirely absent, the insertion of an artificial membrane may be resorted to, provided a preceding funnel test has shown that an improvement in hearing will result therefrom.

Tonybee's artificial membrane, which is intended to close persisting gaps, consists of a round rubber disk of 6–7 mm. in diameter; it is provided with a silver conduction wire.

Lucae devised a complete rubber membrane, with a flexible rubber handle instead of a silver wire.

Lochner draws a fine silver wire or thread through the artificial membrane and inserts the latter through Hinton's tube.

Politzer splits a rubber draining tube in two and attaches a self-made wire handle.

Gruber devised an apparatus for punching out artificial membranes from linen, silk, or rubber, the disks being provided with a silk thread and inserted with a specially devised forceps. The silk thread remains in the auditory meatus and facilitates removal.

Hartmann recommends membranes made of fish-bone fibres, Katz such of celloidin which are made by pouring a 10 per cent. celloidin solution upon a glass or porcelain plate.

Yearsley-Erhard's cotton-wool membrane is very simple and easily applied. A small cotton plug, moistened with sterile vaseline or menthol-glycerin, is pressed against the perforation or stapes region under guidance of the speculum.

Mather advises saturation of the cotton plug with a mixture of carbolic acid, glycerin and alcohol; Baumgarten says that saturation of the plug with cocaine would tend to contract the vessels, reduce the swelling of the mucosa, and thereby improve the vibration of the auricular ossicles.

Urbantschitsch prefers a bland solution (such as salicylic acid 0.2, boric acid 0.3, aq. dest. 20.0, or in ½ per cent. menthol-vaseline), the cotton plug to be saturated and expressed before insertion.

Hassenstein's cotton carrier, 2½ cm. long, which can be left in the external meatus, or Delstanche's cotton brush twisted upon a thin metal wire, can be inserted and removed by the patient himself.

Gomperz advised artificial membranes of vaseline-paraffin, and later such of chemically pure silver foil. The latter are sterilized and introduced in the most favorable position either dry or moistened with a 5 per cent. menthol-vaseline oil. Their advantages consist in being non-irritable, pliable, easily sterilizable, and chemically stable.

Alt uses Gomperz's silver foil, but immerses it in sterile water, from which he fetches it out in the shape of a little lump by means of a boiled forceps. It is then heated over a gas flame until it is nearly water-free, inserted into the tympanic cavity through a speculum, pressed against the promontory wall with burnt cotton, and spread at the promontory with a sterilized sound. By this method the artificial membrane is nearly always tolerated without giving rise to reactions. After the radical operation it may remain in the ear for months, provided the new epidermal layer of the tympanic mucous membrane has been well developed.

According to Hamm's method, a small piece of sterilized gauze, the size of the tympanic gap, is immersed in molten hard paraffin (melting point 113° F.) and inserted into the gap.

Where an artificial membrane cannot be applied owing to the perforation not being marginal, Hammerschlag instils a few drops of vaseline oil into the ear, the head being laterally inclined, and follows this up by air insufflation. The latter causes the liquid to enter the tympanic cavity where it renders service as an artificial membrane. The liquid remains in place for a tolerably long time, owing to capillary attraction in cases where the margin of the tympanic membrane has been preserved.

I am in the habit of using soft rubber caps which the patient can himself insert or remove with a forceps.

The effect of the artificial tympanic membranes upon the hearing ability varies considerably, depending not only upon the degree of the pathologic changes remaining after the middle-ear affection has healed, but also upon the functional sufficiency of the sound-perceiving apparatus. The effect upon one and the same patient varies according to the position of the artificial membrane and the pressure it exercises. Considering that the artificial membrane is really a foreign body, it is intelligible that the ear must get accustomed to it. An initial unfavorable result should, therefore, not prevent a renewal of the attempt. By dint of practice patients often learn to find the correct spot of application themselves.

In any case it is important to keep the patient under close observation for a time, so as to control the functioning of the artificial membrane. Should there be considerable reaction, the membrane is removed until this has disappeared. Slight serous secretions do not necessitate abandoning the membrane, but merely its temporary removal until the secretion has completely disappeared. In the presence of purulent secretion as well as in perforation of Shrapnell's membrane, the use of an artificial tympanic membrane is to be deprecated.

Cotton-wool membranes should be renewed daily at first, but later on may be left undisturbed for days if no reaction manifests itself. Silver appliances, if well tolerated under a suitable system of observation, may be left in place for weeks or months.

The permanent use of artificial tympanic membranes is also to be considered in cases of chronic middle-ear suppuration with continued slight serous or purulent secretion of the middle-ear mucosa, provided the auditory acuity, which is bilaterally considerably impaired, experiences much improvement by their application. Thus, cases of bilateral chronic middle-ear suppuration are by no means rare where children attain sufficient hearing ability to follow the school instruction.

Gomperz's method of insufflating a layer of boric acid has been successful in many cases. It is especially suitable in perforations which expose the stapes in the radical operation. Its improvement upon the

hearing acuity in the presence of a still active secretion, however, will require further tests; should they turn out satisfactory, this method would be preferable in these cases to all kinds of artificial membranes.

The value of artificial tympanic membranes consists not only in improving the hearing ability, but also in offering a certain protection against renewed infections of the middle ear. Nadoleczny and other authors have observed that, in spite of considerable defects, an entirely new membrane was formed under the protective covering of the prothesis.

2. THE SURGICAL FORMS OF CHRONIC MIDDLE-EAR SUPPURATION

These comprise all such cases where, as a rule, surgical interference is required to effect a cure. Some of the lighter cases may certainly result in a cure under conservative measures, while the condition in other cases may be made bearable if treatment is continued for years under incessant control. In the majority of cases, however, the suppuration continues to spread in spite of conservative treatment, with the imminent danger of a sudden or insidious occurrence of a complication.

Complications of middle-ear suppuration resulting from the purulent inflammatory process spreading beyond the normal anatomical area of the middle ear lead to (1) suppuration of the labyrinth, (2) extracranial or (3) endocranial complications.

(a) *Chronic Purulent Osteitis of the Middle Ear*

Chronic purulent inflammation of the temporal bone always occurs under the picture of chronic middle-ear suppuration.

Anatomy.—The secretion in the chronic stage of acute middle-ear suppuration may be confined for a long time to the mucous membrane of the middle ear. In that case the anatomical changes consist in ulceration and granulation of the mucosa, and later in formation of permanent pathological tissues in the middle-ear spaces (pathological ligaments, osteophytes). When the suppuration has led to complete destruction of the mucosal integument at circumscribed places, there will usually occur purulent inflammation and destruction of the bone itself. In the majority of cases such inflammation runs its course under the picture of bone caries, less frequently under that of bone necrosis with sequestration. The result is purulent decomposition or expulsion of the affected parts of the bone. This destructive process is always accompanied by new-formation of bone or a considerable thickening of the bone in the direct vicinity of the pathologic focus (reactive sclerosis, osteosclerosis). Moderate degrees of osteosclerosis are exceedingly frequent in chronic suppuration of bones.

In advanced cases of osteosclerosis all the cavities of the mastoid are obliterated; the bone turns as hard as ivory; the antrum, the tym-

panic cavity, and the external auditory meatus are considerably constricted by dense, compact, pathological deposits of bone. These may finally lead to complete obliteration (osseous atresia) of the antrum, tympanic tubal ostium, external auditory duct, even the tympanic cavity and the labyrinthine window, although the suppuration will persist.

If the destruction of bone spreads more rapidly, extensive abscesses will be formed, filled with stagnating, infectious, and sometimes putrid pus. Continued ulceration involves the danger of a cholesteatoma of the middle ear.

Spreading of osteitis to the auricular ossicles, the attic wall, or tegmen tympani (Fig. 84) leads to sequestration of these parts, exfoliation, or complete suppurative destruction.

Fig. 84.

Sequestration of the tegmen tympani (s) of the left temporal bone in a girl four years old. Death from acute purulent meningitis. *Mai*, internal auditory meatus.

Symptomatology. — The tympanic membrane is perforated; the remnants of it are thickened by scars and often covered with a very thick and resisting epidermis.

The bacteriological examination usually reveals mixed infection. Reliable bacteriological finding can only be obtained on the occasion of surgical operations of the mastoid by sterile removal of pus from the cavities which have previously been closed toward the auditory meatus.

The pathological picture is dominated by the symptom of chronic suppuration. The pus exudes through the auditory meatus, is fetid, and retains its fetid character in spite of energetic antiseptic treatment. This is due to the anatomic conditions which in chronic osteitis prevent free evacuation of the pus, which, being retained in the grooves and furrows of the affected, partly destroyed bone, becomes decomposed. The danger of purulent stagnation is hardly less great in the tympanic cavity and external auditory duct, especially in cases where the mucosa of the duct had already been previously affected by chronic eczema or ulcerations. Cleansing the middle-ear spaces can only imperfectly be done, as, in spite of careful syringing and irrigation with the attic tube in typical cases, pus can again be immediately observed in the otoscopic field by aspiration. This persistent and often threatening retention of pus cannot be overcome by long-continued conservative treatment. The secretion may sometimes be restricted for a few days, but after a short time a copious evacuation will set in without warning, often under the veritable picture of a pus flood.

As a matter of course, the destruction of bone progresses most

rapidly where the bone is thin and tender. Compact osseous substance offers more resistance, and thus it comes to pass that in early dissemination of suppurative osteitis to the mastoid the internal parts of the latter are softened and fluidified at a suprisingly rapid rate, whereas it often requires as much as several years to expel or resorb the lateral ossicles (malleus, incus), which had long before been affected. Similarly, the destruction of the very compact and dense structure of the lateral osseous wall of the superior tympanic cavity and antrum requires a long time. The final result is the complete destruction of the osseous wall and the fistulous perforation of the pus cavity toward the external meatus.

However, it would be quite a mistake to expect to find in each case of osteitis of the middle ear nothing but signs of advancing purulent decomposition of the bone, as there is also early osseous new-formation and condensation of the normal bone, thick layers of connective tissue actually protecting tender bony parts from affection or destruction. In many cases, for instance, the lateral ossicles, which consist of rather massive and compact bones, are partly or completely destroyed at a time when the much tenderer stapes is still intact. This is explained by the fact that the middle wall of the tympanic cavity had been covered in these cases by early deposits of connective tissue and the corners of the labyrinthine windows have been filled with connective tissue. This is facilitated in the stapes region by the normal presence of connective-tissue bridges which need only extending to cover the stapes completely.

If the tube has remained large and permeable, the pus is often evacuated into the pharyngeal cavity, especially in infants, and the fetid odor and taste in the fauces can be perceived by the patients. Abundant evacuation through the tube may simulate suppuration of the accessory nasal cavities or of the sphenoid bones.

The auditory acuity is usually much reduced (V–3½–13½ feet).

The tuning-fork test shows disturbed perception of high sounds aside from the typical signs of an obstacle to sound-conduction as represented by the suppuration of the middle ear. The perception of high sounds is effected by the vestibular section of the cochlea. The new-formation of connective tissue of the internal wall of the tympanic cavity passes on to the blind end of the scala tympani in nearly all cases of middle-ear suppuration. It is through this net of connective tissue that the vibration of the elements of the membranous cochlea is interfered with, leading to degeneration of Corti's organ and the accessory parts of the spiral ganglion and of the cochlear nerves. This is a perfectly typical manifestation, so that the shortened perception of high sounds in the transfusion of the pathological process from the meatus to the internal ear is often a very characteristic sign of chronic middle-ear suppuration.

In other somewhat rarer cases of uncomplicated chronic osteitis of the middle ear, the occurrence of labyrinthine spontaneous nystagmus without vertigo and without equilibrial disturbances must be looked upon as a distant effect, the anatomical explanation of which is similar to that of the cochlear changes.

A comparison of the affected with the healthy mastoid will usually show a diffuse chronic thickening of the periosteal covering, without any tenderness, either spontaneous or on pressure. The maxillary glands, and in neglected cases also the cervical lymph-glands, are enlarged, distinctly palpable, and often painful on pressure. There is often regional headache, with a sensation of fulness and heaviness in the head. The temperature is normal and the general condition in adults usually undisturbed. In infancy continuous fetid suppuration leads to anorexia and disturbance of nutrition, which finally ends in loss of weight, pallor of the mucous membranes, despondency, lassitude, dislike of work, and tendency to fatigue. Children are usually backward in intelligence and make slow progress at school.

Diagnosis.—The diagnosis of osteitis of the middle ear can sometimes be made by the demonstration of the affected bone, especially in infancy. These are cases of exfoliation of the lateral ossicles, destruction of the lateral antrum and attic wall, and sequestration in the antrum or at the fundus of the tympanic cavity. The diagnosis is also supported by the history of spontaneous evacuation of small pieces of bone or by their actual demonstration.

In a large number of cases, however, indirect symptoms have to be resorted to. One of the most reliable is the persistent fetid character of the secretion in spite of energetic long-continued antiseptic treatment of the middle ear. If, notwithstanding cleansing of the middle-ear spaces, rinsing, with subsequent drying up and insufflation of strongly antiseptic powders (iodoform), the pus should retain its fetid properties after five to eight days' treatment, the conclusion of purulent osteitis is justified. Black discoloration of inserted iodoform or xeroform gauze strips within twelve hours, in spite of careful cleansing and drainage of the middle ear, is also a very valuable sign, though not equally reliable. Livid discoloration of the internal end of the auditory duct and ulcerations of the integument in the region of the osseous duct likewise indicate involvement of the bone, and should invariably be responded to by a careful search for osseous fistulæ with the hook sound and attic speculum. Demonstration of an osseous fistula is a sure sign of bone suppuration.

Positive demonstration of purulent osteitis is difficult in cases of slight perforation with sparsely flowing, purulent secretion. This particularly refers to suppuration of the upper tympanic cavity, but the

persistent fetid character of the pus will again be an important sign. Pus retention can in some cases be demonstrated by pus being visible or oozing out upon removal of superficial crusts constricting or occluding the perforation.

Course and Result.—Bone suppuration may be arrested in a small number of cases by conservative treatment, or spontaneously without any treatment whatever. The affected bone is absorbed and heals with a connective-tissue scar. There remain no defects except those mentioned as signs of a previous chronic suppurative osteitis, consisting in condensation of the bone in the immediate vicinity and connective-tissue scars of the medial wall of the tympanic cavity or pathologic connective-tissue bridges in the cavity itself. In the majority of cases, however, the gap in the tympanic membrane will persist, owing to its margin becoming invested with epithelium. In many cases restoration does not take place by normal tympanic tissue, but by more or less thin scar tissue, connective-tissue scars, or calcified connective tissue (Plate VIII). It is not surprising, therefore, that in most cases the hearing distance is permanently reduced, and good functional sufficiency after chronic osteitis constitutes a rare occurrence.

In about 80 per cent. of all cases of chronic osteitis of the middle ear, suppuration defies all conservative treatment. Middle-ear changes, which must be looked upon as initial signs of healing and are demonstrable in nearly all cases, are accompanied by progressive dissemination of the bone suppuration. The progress in healing, however, does not keep step with the progress of the suppuration, the result in the most favorable cases being suppuration continued for life within the anatomical borders of the middle ear. Gradual degeneration of the nerve-terminals of the internal auditory canal finally leads to the highest degree of partial or to complete deafness. In other cases the suppuration of the bone spreads to the surrounding parts, so that in the end distant parts of the temporal bone become affected (mastoiditis, ulceration of the temporal squama and zygomatic process, suppuration of the labyrinth) or a perforation occurs with a descending abscess. The ulceration may also spread to the cranial cavity, either direct or by the route of the mastoid or labyrinth. This involves an endocranial otitic affection.

Treatment.—When the bone suppuration is superficial, well demarcated, and confined to the tympanic cavity, conservative treatment may be successful, provided a spreading of the osseous process can be prevented while the treatment is going on. This particularly refers to cases where the lateral osseous attic wall, the osseous trabeculæ of the hypotympanum, or the auricular ossicles are affected. In the other cases nothing but surgical interference will effect a cure.

(b) *Cholesteatoma of the Middle Ear*

Anatomy.—Primary and secondary cholesteatomata are to be distinguished. The former belongs to the group of congenital tumors and owes its existence to displaced embryonal epidermal germs which independently continue to grow. Primary cholesteatoma in the ear has only been observed in very rare cases as a congenital cholesteatoma of the dura mater which has spread to the ear by gradual increase in size.

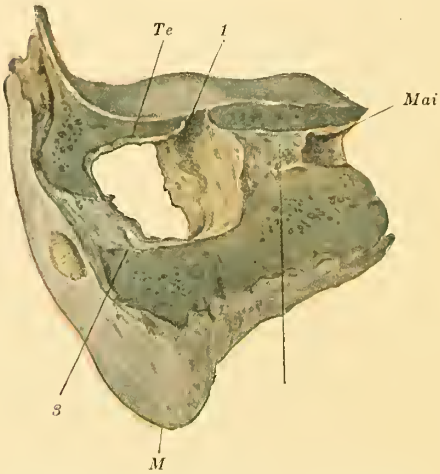
Secondary cholesteatoma of the middle ear (Figs. 85–88) demands great clinical interest, as it never occurs except in the wake of chronic middle-ear suppuration. It may be briefly termed cholesteatoma, and its evolution takes place in the following manner: The epidermal layer of the external auditory duct gradually grows into the middle ear, either direct or by the aid of misplaced isolated epidermal islands, while the middle-ear suppuration continues. Owing to the presence of an otorrhœa, which may have existed for many years, the epidermal layer of the auditory duct, especially at its internal end, develops pathologic proliferation. Penetration of this rapidly proliferating epidermis into the middle-ear space is facilitated and rendered possible by (1) marginal perforation or complete destruction of the tympanic membrane, (2) perforation of Shrapnell's membrane and defects of the lateral attic and antrum wall.

The fact of the epidermis advancing into the middle-ear spaces does not necessarily lead to the formation of a cholesteatoma; in fact, as the epidermis advances into the middle ear, it may occasionally produce a cure of a chronic middle-ear suppuration by furnishing the required epidermal material. This is particularly the case in simple chronic middle-ear suppuration where the osseous parts of the middle ear have been preserved. If, however, the bone is affected, the penetrating epidermis will soon degenerate into pathologic proliferation, owing to the continuation of the suppuration. In the course of this process, isolated epidermal groups may be displaced underneath the surface between the granulation wall and the bone, where they continue to grow independently. The unavoidable consequence is the formation of epidermal balls, consisting of concentric epidermal layers (Figs. 86, 87). Their central parts are composed of macerated cell masses, profuse quantities of detritus, micro-organisms, fat crystals, and cholesterin crystals. The superficial layers (Figs. 85, 88) of the cholesteatoma (matrix) are more intensely proliferated. The development of the superficial epidermal layer is so intense that the soft parts as well as the bone are completely destroyed as the cholesteatoma increases in size. The organic substance of the bone perishes; the inorganic salts are deposited in the nearest bone in the vicinity. This bone, being thus dis-

tinguished by a superabundance of lime, joins in the proliferation. As a consequence, a very dense osseous layer, as hard as ivory, forms around the cholesteatoma, and even the macerated specimen shows the smooth demarcation of the cavity (Fig. 88), as distinguished from the furrowed, ribbed bone cavity after caries and sequestration.

Renewed infection of the cholesteatoma, which usually occurs during an acute exacerbation of chronic middle-ear suppuration, is the cause of exceedingly dangerous, acute putrefaction of the cholesteatoma (Fig. 85).

Fig. 85.



Cholesteatoma of the middle ear and labyrinth. Right temporal bone. The central part of the cholesteatoma is decomposed and spontaneously expelled. The peripheral layers and the matrix are firmly attached and sharply contrast against the green color of the bone by their yellow-gray color and mother-of-pearl lustre. The contrasting colors of the specimen are the result of preservation in Müller's fluid and subsequent treatment with alcohol. The cut surface corresponds to a vertical section through the temporal bone in the region of the tympanic cavity near the antrum. The cholesteatoma has led to three fistulæ in different directions: (1) fistula through the tegmen tympani to the dura of the middle cranial fossa; (2) complete suppurative destruction of the labyrinth with fistulous eruption into the internal auditory duct (*Mai*); (3) fistulous eruption through the lateral wall of the mastoid process (*M*). Observe the condensation of the entire preserved part of the bone. (Natural size.) Boy thirteen years old. Death from diffuse purulent meningitis.

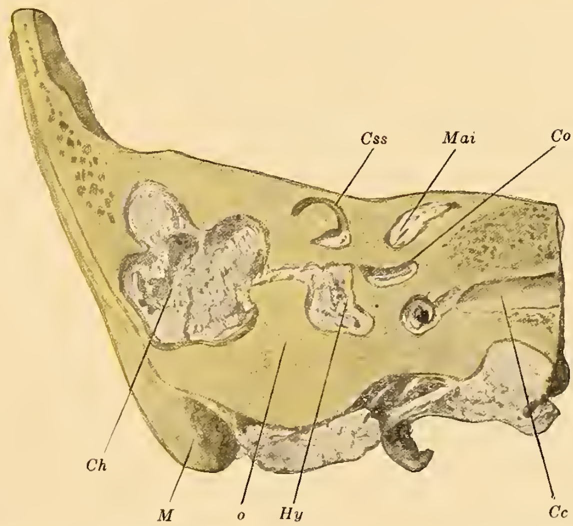
Symptoms. — In typical cases there is a profuse discharge of highly fetid pus, permeated with white crumbs and grayish-yellow epidermal masses. If the cholesteatoma has been destroyed by acute putrefaction, large numbers of particles are removed through the external meatus. In other cases particles of the cholesteatoma can be recognized in the otoscopic picture by their grayish-white color and the stratified structure of the tumor. In some cases cholesteatoma gives rise to no special manifestations; in the others the otoscopic picture shows an apparently healed middle-ear suppuration, and nothing but the periodical return of the suppuration, as elicited by the history, points to the continuance of the pathological process. In cholesteatoma of the antrum the posterior wall of the auditory duct has considerably descended; if the attic wall is destroyed, a cholesteatoma of the attic may be freely visible in the otoscope. Should the cholesteatoma have grown beyond the borders of the middle ear and led to complications, it is the clinical manifestations of the latter which suddenly spring into view. For the symptomatology see the corresponding chapter (pp. 234–275).

Diagnosis.—The diagnosis of cholesteatoma may in some cases be made from the above-described peculiarities of the suppuration or from the otoscopic findings. Defects of the osseous wall of the auditory duct

and signs of mastoiditis in chronic middle-ear suppuration likewise point to cholesteatoma. An exact clinical diagnosis, however, can be made only by a microscopic examination of the pus taken from the middle ear.

For this purpose the external auditory duct is carefully cleansed with benzine and a 5 per cent. perhydrol solution, special attention being paid to the cleansing of the angle and fundus. Some pus is expelled from the middle ear by irrigation with the aid of an attic tube, spread on a slide in physiological salt solution, and examined while fresh. With medium microscopic enlargement the characteristic cholesterin crystals

Fig. 86.



Frontal longitudinal section through the right temporal bone. Cholesteatoma of the middle ear and labyrinth. Posterior half of the section. (Natural size.) A cholesteatoma about the size of a cherry fills the upper part of the mastoid process (*M*). The bone in the vicinity (*o*) is condensed. Cholesteatomatous masses fill the hypotympanum (*Hy*) and have advanced into the labyrinth (*Css*, *Co*). *Css*, canalis semicircularis superior; *Co*, cochlea; *Mai*, meatus auditorius internus; *Cc*, canalis caroticus.

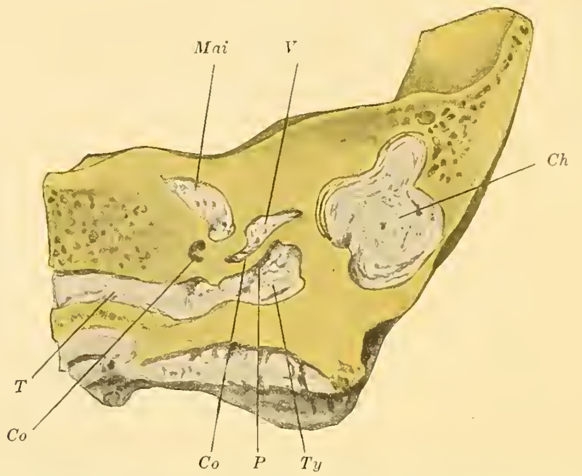
will be found, which may number up to 100 in one field of vision, especially if the cholesteatoma has putrefied; in other cases only a few crystals may be present.

The cholesterin test may prove negative in spite of the presence of a cholesteatoma if the latter is covered by granulations, or the auditory duct is occluded by polypi, and in cases where the irrigation fluid cannot reach the cholesteatoma owing to pathological changes in the external or middle ear.

Course.—In most untreated cases the course is unfavorable, owing to the constant increase in size as well as to the possibility of sudden putrefaction. The latter contingency is fraught with special danger if the cholesteatoma has already exposed the dura or advanced up to the cavities of the labyrinth. Acute putrefaction of a cholesteatoma is

responsible for at least 60 per cent. of all endocranial otogenous affections and for many cases of otogenic paralysis of the facial nerve and suppuration of the labyrinth.

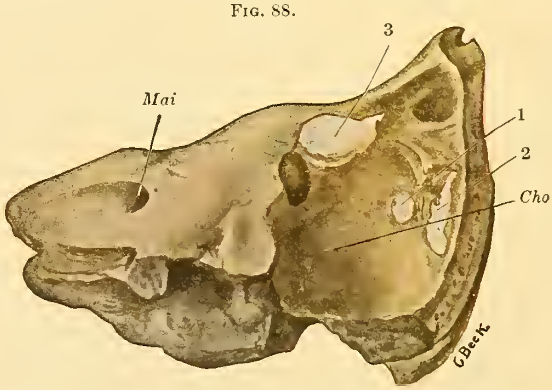
Fig. 87.



Frontal longitudinal section through the right temporal bone. Cholesteatoma of the middle ear and labyrinth. Anterior half of section. (Natural size.) *T*, auditory canal; *Ty*, tympanic cavity; *V*, vestibule; *Ch*, Mastoid cells filled with cholesteatoma; *P*, promontory; *Co*, cochlea; *Mai*, meatus auditorius internus.

The subjective and objective manifestations may be reduced to bearable proportions by conservative treatment in a number of cases, but complete and permanent recovery is only exceptionally possible by conservative treatment.

Fig. 88.



Cholesteatoma cavity (*Cho*) of the mastoid process. Right temporal bone seen from the posterior surface of the petrous bone (*Mai*, meatus acusticus internus). Observe the mother-of-pearl lustre of the smooth matrix of the cholesteatoma (*Cho*). 1 and 2 are fistulous perforations through the lateral wall of the mastoid; 3 is a fistulous opening of the middle cranial fossa. The cavity is wide open toward the posterior cranial fossa owing to the large osseous defect. Death occurred from infectious sinus thrombosis and pyæmia. (Natural size.)

The surgical treatment consists in the radical operation. Attico-antrotomy and plastic of the auditory duct are done in well-demarcated cholesteatoma of the attic or antrum where there is good hearing acuity.

Indications.—A relative indication for operation exists in all cases of established cholesteatoma of the middle ear with normal or at least good hearing acuity of the opposite side. Operation is contraindicated in partial or complete deafness of the other ear and in constitutional affections.

An absolute indication for operation exists in cases where the cholesteatoma has led to paralysis of the facial nerve and where there are clinical signs pointing to erosion of the capsule of the labyrinth or to exposure of the dura mater or sinus.

(c) *Chronic Middle-ear Suppuration Complicated by Ulceration and Stenosis of the External Auditory Canal*

This is not amenable to conservative treatment. Formation of fistulæ in the osseous external duct is usually observed when the osseous wall has been destroyed by the proliferating cholesteatoma. Ulceration of the posteriosuperior part of the auditory duct always gives rise to the suspicion of a fistula which can be verified by the sound. X-ray photography also renders good service in some cases.

Removal of granulations and polypi from the external duct is usually useless, as their recrudescence cannot be prevented in spite of continued conservative treatment. Operative treatment is even more indicated, as it is just purulent osteitis of the middle ear which leads to ulceration of the auditory duct.

(d) *Chronic Middle-ear Suppuration Complicated by Purulent Mastoiditis*

Purulent inflammation of the mastoid process sometimes develops insidiously in the course of a chronic middle-ear suppuration. Periostitic, painless thickening of the covers of the soft mastoid parts is a prodromal symptom. Such forms of inflammation as resemble acute purulent mastoiditis in the course of acute middle-ear inflammation will only occur if there has been sequestration of the mastoid or acute putrefaction of the antrum and mastoid in the course of chronic middle-ear suppuration.

In these cases the symptoms of mastoiditis are caused by accumulation of pus in the mastoid (chronic empyema), by sequestration or ulceration of a cholesteatoma; they are often complicated by sinus thrombosis and pachymeningitis. Nothing but timely radical operation offers a chance for recovery.

Fistulous perforation into the osseous external duct will occur after mastoiditis or suppuration of the antrum has existed for some time, especially in the presence of a cholesteatoma. Destruction of the osseous wall continues uninterrupted, the final result being complete destruction of the posterior and superior parts (natural radical operation). These

cases may recover spontaneously after a radical operation, or under conservative treatment directed to the re-formation of epidermis in the middle-ear spaces, but they are exceptions. In all other cases the middle-ear suppuration can only be arrested and vital complications prevented by timely radical operation.

(e) *Chronic Suppuration of the Upper Tympanic Cavity* (*Epitympanum or Attic*). *Chronic Suppuration of the Antrum*

Owing to the topographical position of the auricular ossicles, their muscles and ligaments, the meso- and epitympanum normally communicate by fissures. The communication between the meso- and epitympanum may be completely interrupted by the formation of abnormal ligaments and abnormal connective-tissue layers at the point of communication, owing to catarrhal affections of the middle ear. The lowest part of the epitympanum in the normal upright position of the head is formed by Shrapnell's membrane, and the small aperture of the latter is the only way of escape for the pus formed in suppurative inflammation owing to interrupted communication between the meso- and epitympanum. Thus epitympanic suppuration is one of the most dangerous forms of middle-ear suppuration, and tends to early chronicity and failure of conservative treatment. Furthermore, perforation of Shrapnell's membrane favors the formation of a cholesteatoma of the middle ear, and, owing to the smallness of the membrane, the perforation soon extends to the osseous margin. A cholesteatoma of the attic or antrum may easily develop, considering that Shrapnell's membrane has a thick epidermal layer of its own with papillary rudiments, and that a pathological proliferation of the epidermis must unavoidably cause the latter to grow into the upper tympanic cavity or into the antrum.

Symptoms.—Suppuration of the attic and antrum is characterized by the insidious nature of its onset and course. There is no pain whatever in the incipient stages. If the hearing acuity is but slightly impaired and that of the other ear normal, the affection may remain undetected for a long time, until there is suddenly a purulent secretion from the external meatus. Further dissemination of the purulent inflammation may cause earache and headache. Extension of the process to the chain of auricular ossicles usually injures the hearing acuity to a considerable extent. In pus retention there is headache in the temporal region of the affected side, susceptibility to percussion in the temporal region, and sometimes moderate elevation of temperature.

The conditions of the tympanic membrane cannot be mistaken in most cases (Fig. 89). The seat of the affection is usually marked by a small shiny drop of fluid covering the region of Shrapnell's membrane. On removing the secretion with cotton tips, a slightly

granulated surface will be seen in some cases, a granulation-polypus growing out of the perforation gap in others. Where the secretion is slight, the perforation may be closed by a brown ceruminal crust, on removal of which pus becomes visible. It is certainly necessary in all cases of affections of the ear to establish carefully the exact condition of the membrana flaccida. If the lateral osseous wall is destroyed by ulceration, the otoscope will reveal a more or less extensive gap, through which the attic and the parts of the auricular ossicles situated therein become plainly visible (Fig. 90, 1). If the auricular ossicles are destroyed, the attic is either empty or replete with granulations. In these cases atticoscopy in conjunction with the attic speculum, recommended by Urbantschitsch, renders excellent service in the examination of the attic and antrum.

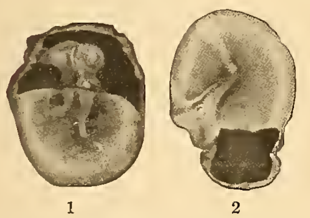
FIG. 89.

Tympanic membrane in chronic epitympanic middle-ear suppuration. Perforation of the right membrane of Shrapnell.

The parts of the auricular ossicles situated in the attic are either primarily affected, or secondarily by the spreading of the attic suppuration.

Pus retention in the attic and antrum not infrequently leads to descent of the posterior wall of the auditory duct. Besides, fetid pus may be seen to exude shortly after the middle ear has been cleansed, although sometimes it only appears upon aspiration with Siegle's irrigation apparatus.

FIG. 90.



1. Chronic attic suppuration with destruction of the lateral attic wall. 2. Chronic antrum suppuration with destruction of the lateral antrum wall.

The upper tympanic cavity is separated from the middle cranial fossa by the tegmen tympani, and pus retention in the former favors spreading of the purulent inflammation to the latter.

Tuberculosis of the middle ear (q.v.) begins in a fair number of cases under the picture of torpid suppuration of the attic or antrum.

Suppuration of the antrum as well as of the attic often sets in without any symptoms. The perforation may be situated in the posterosuperior quadrant (Plate VIII, 12; Fig. 90, 2) or in the auditory duct itself. In the latter case it may be difficult to recognize it in direct otoscopy, and its presence should therefore be carefully established by a curved sound and aspiration of purulent secretion with Siegle's irrigation apparatus.

Treatment.—Conservative treatment leads to recovery in some cases where the ulceration is well demarcated. In caries of the auricular ossicles, ossiculectomy is often effective, provided the middle-ear spaces are otherwise normal. In all other cases radical operation is indicated, especially in the presence of a cholesteatoma of the

attic or antrum. Where there is good hearing acuity and a perfectly normal meso- and hypotympanum, attico-antrotomy may suffice.

OPERATIVE TREATMENT OF THE SURGICAL FORMS OF CHRONIC MIDDLE-EAR SUPPURATION.

1. Excision of Malleus and Incus (Ossiculectomy)

The operation is done under local injection anæsthesia. The malleus is circumcised with a narrow little knife and slowly mobilized with a small forceps. When sufficiently mobilized, it is firmly grasped with an ear-forceps or the Sexton forceps (double-hook forceps), pulled out downward, and extracted. Delstanche's circular knife cannot be recommended for this operation, as it easily fractures the crura of the stapes or may dislocate the stapes itself. Should the malleus and incus be united either by pathologic connective-tissue layers or bone, both are extracted together. In other cases it will be necessary first to draw the incus downward with the forceps or to deflect the same from the antrum to the mesotympanum with the aid of small sharp spoons. This operation requires practice and caution, and previous experience. A careful examination with the probe or attic speculum should have positively established the normal site of the incus. In many cases of antrum suppuration the incus is destroyed, and nothing but malleus and stapes remain.

Rough procedure in extracting the incus may lead to injury of the osseous facialis canal and paralysis of the facial nerve, and even luxation of the stapes. It need not be specially mentioned that the auricular ossicles must not be extracted if there are signs of impending labyrinth affection. The wound caused by the extraction has to be carefully tamponed with iodoform wicks and bandaged. If the patient is free from fever and pain, the bandage remains undisturbed for four days. On the fourth day the wicks are pulled forward one by one, shortened by half their length, and completely removed on the following day.

2. The Various Methods of Performing the Radical Operation. (Operative Exposure of the Middle-ear Spaces)

The object of the operative exposure of the middle-ear spaces is to unite the various cavities of the external and middle ear into one uniform traumatic cavity without sharp projections, deep sinuses, or angles, so far as such may be possible.

In those cases of chronic middle-ear suppuration in which the mastoid process does not show any signs of suppurative inflammation it will be sufficient to unite the antrum, hypo-, meso-, and epitympanum and the external auditory canal by removing the various septa. The

mastoid cells are opened and removed with the chisel only as far as may be necessary for opening of antrum and removal of the posterior osseous wall of the auditory duct (conservative exposure of the middle-ear spaces; synonyms: radical operation after Zaufal; radical operation after Stacke).

In the radical operation performed by Zaufal the antrum is exposed with the chisel, starting from the antrum triangle (Fig. 79), after which the presenting posterior osseous wall of the auditory duct is removed. In the radical operation recommended by Stacke, which is but seldom resorted to in children, the chisel work commences with the removal of

Fig. 91.

Topography of the middle ear after exposure of the middle-ear spaces and opening of the canalis facialis and of the lateral semicircular duct. (Left temporal bone, $\frac{2}{3}$ natural size.) *Eal*, eminentia arcuata lateralis (opened); *M*, mastoid process; *Pro*, promontory; *Ss*, sulcus sigmoideus; *Fc*, fenestra cochleæ; *Cvii*, canalis facialis.

the posterosuperior osseous wall of the auditory duct. In every conservative exposure the middle-ear spaces are to be opened outwardly far enough to prevent any retention of secretion or pocket formation. Special care should therefore be taken to remove completely the lateral osseous attic wall (Fig. 91).

The osseous crest, demarcating the hypotympanum, is likewise completely removed, so that the fundus of the osseous external auditory duct may continue into the osseous fundus of the tympanic cavity on precisely the same level. With the object of obliterating the tube, its mucous membrane is curetted. Whenever the tympanic cavity becomes pointedly funnel-shaped toward the tubal opening, it would be advantageous to attenuate or remove the anterior part of the tympanic membrane with the chisel or the bone-forceps. This will effectually serve to prevent the formation of a fistula or funnel at the tubal opening, at the same time favoring the formation of a solid cicatricial occlu-

sion of the tube. The posterior wall of the osseous auditory duct is opened up to the facial canal, except that a slightly protruding convex crest remains (Fig. 92). Sufficient removal is attained if the upper end of the facial region does not lie higher than the prominence of the lateral semicircular duct. The basal inferior part, however, should only be removed far enough to allow the promontory to be seen at a lateral aspect. On the other hand, the window of the cochlea should be invisible in the position the examiner now occupies. Should it be present in the field of vision, it would indicate that too much bone has been chiselled away in the facial region and the facial nerve has been endangered.

The exposed cavities are cleansed with sharp spoons and curetted, abstaining of course from any curettage whatever of the lateral wall of the labyrinth. Rough mopping and any force whatever should be avoided; any slight hemorrhage, even though disturbing, is best checked by the insertion of adrenalin tips rather than by repeated mopping of the traumatic cavity, as the latter proceeding may endanger the stapes.

In chronic cases, where the hollow cavities of the mastoid process are involved in the suppurative osseous affection, all the spaces of the mastoid process are to be opened as well (Fig. 91). This is the typical radical operation or radical exposure of the middle-ear spaces (synonym: radical operation after Kuester-Bergmann). In this operation we start from the mastoid triangle (Fig. 79), carry out the exposure after the type of the conservative method of operation above described, after which the osseous contents of the mastoid itself are removed. But whether the exposure has been performed according to the conservative or radical method, it is important that the smoothing out of the osseous parts toward the middle cranial fossa and the sigmoid sinus be reserved for the final act of the operation, so as to obviate the necessity of carrying through the entire ear operation with the exposed dura of the middle cranial fossa or with the exposed and possibly bleeding sinus. Accidents of this kind cannot be prevented by the most experienced.

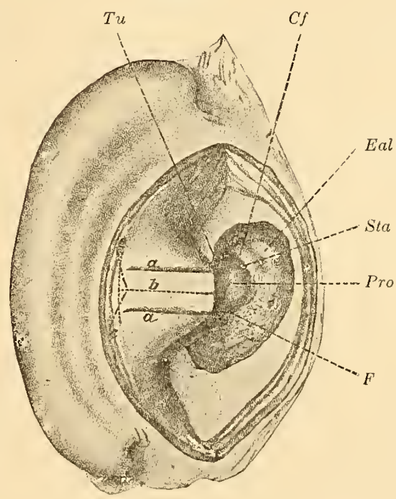
Owing to the removal of the posterior osseous wall of the auditory canal, the membranous canal remains in the shape of a cartilagino-membranous soft tube, which protrudes into the traumatic cavity and is now, after the operative exposure of the middle-ear spaces, inserted merely at the tympanic membrane along the anterior wall of the auditory duct. The fate of such a soft ear would be suppurative disintegration in the course of the traumatic process, ending with cicatricial stricture. It is necessary, therefore, to supply the membranous auditory duct with a plastic support after every radical operation.

The incisions necessary to supply the plastic support may vary (Fig. 92). Panse applies a T-shaped incision. If the longitudinal incision were not made in the middle, but toward the upper or lower end

of the vertical incision, one single flap would be obtained, the base of which would be situated either upward or downward. On the other hand, in Panse's plastic operation there will be an upper and a lower flap. In these three methods there will be a very spacious auditory meatus, which, however, is demarcated by a traumatic line at its posterior half. This may give rise to difficulties in the after-treatment and lead to a stricture of the auditory meatus. In cases where the vertical incision lies very far laterally, exposing the cartilage, there will also be danger of suppurative perichondritis.

It is not surprising, therefore, that these methods have undergone modifications. Brühl leaves a square flap at the auditory meatus, Siebenmann (Fig. 92) and Neumann a triangular flap which can be drawn backward by a catgut suture. In this way the remaining auditory meatus is entirely surrounded by epidermis, so that there can be no question of perichondritis, as any cartilage which should be exposed will be at once plastically covered.

Fig. 92.



View of the operative field after complete conservative exposure of the middle-ear spaces (radical operation after Zaufal). (Left ear, $\frac{3}{4}$ natural size.) *Tu*, tympanic tubal opening; *Cf*, canalis facialis; *Eal*, eminentia arcuata lateralis; *Pro*, promontory; *F*, smoothed-out facialis spur; *aa*, incision after Koerner's plastic; *b*, incision after Panse; *c*, incision after Siebenmann; *Sta*, stapes.

The methods so far mentioned are principally devoted to the formation of a convenient auditory meatus. But the resulting flaps are relatively short, with a broad base, which can advance but little into the operative cavity. The healing of the operative trauma occurs by the bone being gradually covered with epidermal epithelium, while the osseous cavity itself will persist. The details of the healing process will again be referred to, and at this juncture it may merely be mentioned that it is important to advance the epidermal flaps from the first as far as possible into the osseous cavity, since it is from these flaps that the growth of the epidermal layer leading to a cure will take its start.

From this stand-point we ought to try to obtain very long flaps with a narrow base. Koerner's plastic operation (Fig. 92) is the closest approach to this demand, but the long tongue-shaped flap is suitable only for cases in which healing without reaction can be foreseen during the operation and the retro-auricular wound immediately closed up to a small aperture at the lower traumatic angle. For the other cases Passow's plastic operation is best adapted, especially where the traumatic cavities are large, in cases of an exposed posterior cranial fossa, in cases

of sinus thrombosis, and in cases of total suppuration of the mastoid process. With this operation the flaps are certain to remain attached to the traumatic cavity, while pocket formation is excluded.

If the auditory canal was ample, we shall confine ourselves to the skin of the canal and not incise beyond the meatus. In the opposite case there is danger that after closure of the retro-auricular wound—which we should always endeavor to effect—the auditory meatus will not be sufficient for the after-treatment or for the necessary control of the traumatic cavity. In cases of this kind the auditory meatus may be enlarged by prolonging the incisions into the cymba conchæ or, as the case may be, continue the lateral incisions beyond the normal auditory meatus. On the other hand, too large a meatus will later result in a considerable deformity, and this contingency should be avoided, as it is entirely unnecessary. For this reason, I am unable to endorse the proposition of some authors to incise the cartilage of the auditory duct and the concha in order to obtain an exaggerated meatus.

The fate of the retro-auricular traumatic cavity depends upon the peculiarities of each case. Where an entirely uncomplicated healing process can be foreseen, the retro-auricular wound is closed down to the lower traumatic angle immediately after the operation. Mitchell's clamps are best for this purpose. Only a few wicks from the antrum region are conducted outward through the lower angle. These are cases which previous to the operation did not show any signs of the surrounding parts being affected and in which the operation confirmed that the suppurative inflammation had been entirely confined to the middle ear.

In some clinically uncomplicated cases, however, it may be seen at the operation that the bone is diseased up to the dura, most frequently at the middle cranial fossa, so that the dura, although not affected, lies exposed in the operative cavity. In cases of this kind as well as in cases where a venous sinus has been exposed, I am in the habit of narrowing the retro-auricular skin wound by suture clamps, but allowing a bundle of wicks about the thickness of a little finger to protrude on the outside. In cases complicated by endocranial or labyrinthine involvement the retro-auricular suture had better be avoided; in extensive incisions (in cases of sinus thrombosis) the skin flaps can only be approximated by a bridle suture.

After-Treatment Following Radical Operation.—The first bandage covers half the head, including the neck. In cases which run a normal course the bandage is changed on the sixth day. All gauze layers are removed to the wicks; the latter are pulled out by about 2 cm. and shortened.

After that the bandage is changed every second day, and wicks are gradually shortened.

In wounds of medium size all the wicks are usually removed with the fourth change of bandage. The wicks are to be pulled out very carefully, each one being caught separately with the forceps and handled with the greatest care. In this way the patient is saved unnecessary pain, and the cavity will not be subjected to any unnecessary trauma. If the bandages have been changed with the necessary care, there will be no hemorrhage as the wicks are being removed.

In the further course the frequency of the changes will depend upon the quantity of the traumatic secretion, which differs considerably in various cases. The bandage must not be allowed to remain after it is no longer effective. The change of bandage, if done at the proper time, will show the outer gauze layers partly saturated by the secretion. After the strips have been removed, the traumatic cavity should be free from pus. If the cavity as well as the lateral surface of the concha and the parotid region are covered with secretion, it is a sign that the bandage has remained too long in place and should in future be renewed more frequently. The largest quantity of secretion is discharged in the third week after the operation, and at about that period it will nearly always be necessary to renew the bandage daily.

In cases of anæmia, diabetes, scrofulosis, or tuberculosis the quantity of secretion is sometimes exceedingly large and may require a change of bandage several times in a day.

An unpleasant smell of the secretion is an indication of carious or necrotic particles of bone having remained behind; sometimes it is a sign of an insufficient operation. Fetid secretion may sometimes be caused by particles of bone which have been chiselled off and retained in the cavity. If the tendon of the sternocleidomastoid muscle has not been properly removed when exposing the mastoid process, it may happen that particles of bone remain attached to the tendons, become necrotic, and lead to fetid secretion. After they have been removed or become detached spontaneously, the fetid character of the secretion will rapidly lose itself.

If the trauma runs a normal course, no exposed bone will be visible at the beginning of the fourth week. The cavity is completely invested with granulations, and a bluish-gray edge indicates the growing epithelium.[1] At this stage the rapid covering of the granulating traumatic surface with epidermal epithelium must be facilitated by treatment. Care should be taken to prevent uneven growth of the granulations. Such a contingency is nearly always caused by stagnation of the secretion, and the patient should therefore be instructed to renew the gauze strips himself several times daily, and to remove the secretion mornings and evenings by instilling into the ear a few drops of peroxide of hydrogen.

[1] In cases where the healing takes place without reaction, transplantation of the epidermis may be resorted to according to Politzer's method.

The growth of the epidermis is aided by insertion of cotton plugs saturated with alcohol. Where alcohol does not cause pain, hyperæmia, or acute increase of the secretion, patients may instil the alcohol themselves. For this purpose either a mixture of perhydrol and alcohol may be used, or perhydrol may be instilled in the morning and alcohol in the evening.

For tamponading the traumatic cavity I use sterile gauze bandages 12 feet long and 1 inch wide. The strip in the external meatus may be renewed by the patient himself as often as necessary, observing the greatest cleanliness.

Under this treatment the epithelium may grow completely in 6–8 weeks, but deviations from this ideal course are of such frequent occurrence that they should be specially discussed.

If cicatrization of the tympanic tubal end does not take place, and there is no suppurative secretion in the region of the tube, membranous occlusion of the antero-inferior part of the tympanic cavity may be expected. In these cases I insert the gauze strip merely toward the antrum,—*i.e.*, into the back part of the tympanic cavity,—so that the spontaneous epithelial proliferation in the anterior part of the cavity may not be disturbed.

If, on the other hand, there should be an accumulation of suppurative secretion in the tubal region, the antero-inferior part of the cavity must be kept permeable by careful tamponade, and the formation of a septum be prevented. The tampons are renewed once a day, except when the secretion is very considerable, when they should be renewed 2–4 times daily. Cauterization with absolute alcohol, chromic acid, and insertion of scarlet red (Stein) once a week renders good service. As the epithelium grows, I assist the process by applying sunlight baths in summer, at other seasons lamplight; in office practice I insert small, cold electric lamps into the cavity, where they are allowed to remain for 5 minutes. In very obstinate cases, where obliteration of the tubal ostium will not take place, I apply curettage to the tympanic tubal end under local injection anæsthesia. Development of a septum would be dangerous in these cases, as the secretion would stagnate behind the septum and, after an apparent cure, break out afresh. Later, insertion of 5 per cent. airol-vaseline or 5–10 per cent. cycloform ointment (for traumatic pain) is often beneficial.

The radical operation may also result in a cure with formation of a membrane which is nearly always situated on the level of the former remnant of the tympanic membrane and which evidently has its starting-point at the remnants of the inferior margin of the tympanic membrane. In many cases this membrane is inserted along the facial region; in others it runs to the backward margin of the traumatic cavity,

closing not only the tympanic cavity, but laterally the antrum region as well. The thickness of the membrane usually corresponds with the thickness of the tympanic membrane, but the fibrous layers show no regular arrangement. The membrane itself is perfectly smooth and covered at its lateral surface with epidermal epithelium, which has sometimes a metallic lustre. The middle-air spaces which are closed through the membrane contain air, and the ventilation occurs as in normal conditions and by way of the tube in the act of swallowing.

In some cases the formation of the epithelium and cicatricial occlusion of the tube may proceed without difficulty, but the granulations themselves remain thick and massy and are finally completely changed into connective tissue. This kind of wound, when healed, presents the otoscopic picture of a small hollow ball. The characteristic contour of the internal wall of the tympanic cavity is completely covered by the thick connective-tissue layer.

According to the way a radical operation heals, the cases may be divided into the following four groups:

(1) Cases with formation of epithelium, perfect preservation of the osseous cavity, and obliteration of the tympanic tubal opening.

(2) Cases with a membranous tubal septum.

(3) Cases with a membranous occlusion of the middle-ear spaces and permeable tube.

(4) Cases with connective-tissue atresia of the middle ear.

All these results are practically of equal value and permanent, provided the patient's condition of nutrition is favorable.

The membranous occlusion of the middle-ear spaces has the advantage of completely protecting the latter against injury in a normal and anatomical manner from without. There is, however, the possibility of the patient contracting a typical acute inflammation of the middle ear if an acute infection of the nasopharyngeal space should set in, and especially if chronic suppurative conditions should create a predisposition for that affection.

The physiological evacuation of the secretion of the auditory glands is usually completely arrested, owing to the external canal having entirely lost its tubal form and curvature through the plastic operation, so that one must be prepared for accumulation of cerumen in the operative cavity after every radical operation. If the middle wall of the tympanic cavity is exposed, pressure of these masses upon the wall of the labyrinth will cause headache and a sensation of fulness and heaviness in the ear, sometimes with objective signs of irritation of the labyrinth (spontaneous nystagmus, vertigo).

In membranous occlusion of the middle ear this danger does not exist, and in this respect the result is preferable.

It is a matter of importance that the postoperative hearing acuity is the same in all four methods of healing.

The epidermal epithelium investing the operative cavity is rapidly renewed during the first few months following operation, as scabs and crusts will be formed. If these are allowed to remain in the ear, maceration of the epithelial layer underneath and circumscribed ulceration may take place. In order to prevent this, patients are instructed to wear short sterile gauze strips after the wound has healed, and to renew the same daily. With their removal, the loose crusts are likewise removed. Besides, during the first year after operation the patient should attend at the physician's office one or twice a month and have the epidermis removed with the forceps should the same have been loosened, so that the cavity may always be covered with smooth, gray, glistening epidermis.

The complete resection of the mastoid process means a rather considerable loss, and if it has been necessary to keep the retro-auricular wound open for a long time, it will be impossible to prevent the persistence of a retro-auricular aperture for some time, which is separated by an epithelial margin. Such an aperture may be closed by a direct suture up to 2–4 weeks after the operation. Later, however, the suture would cut through, and the aperture would reappear. All the methods for closing the retro-auricular gap are intended to provide a double layer of skin, one epidermal surface being turned against the operative cavity and the other toward the outside.

Von Mosetig forms the lower flap from the region immediately below the mastoid process and closes the incision by sutures. Therefore, a new scar reaching to the neck is added to the operative scar, so that from a cosmetic point of view this method is not to be recommended. The disadvantage of Passow's method consists in both sutures being immediately above one another, so that they will easily cut through, like a simple suture. The method employed by myself has the great advantage of the two sutures not lying immediately above one another, so that each of them is supported by the skin flap above and below.

3. *Attico-antrotomy*

In cases of chronic suppuration of the attic and antrum with well-preserved hearing acuity, the typical radical operation may be replaced by the much less severe attico-antrotomy. In this procedure the antrum is exposed from the mastoid process as in antrotomy. The lateral attic wall is removed until the junction of the malleus and incus becomes visible. The frame and the pars tensa of the tympanic membrane are preserved. After the performance of attico-antrotomy, Koerner's plastic operation of the auditory duct is done and the tongue-shaped flap pushed

toward the posterior wall of the antrum. The retro-auricular wound is closed down to the lower angle, a thin drainage strip being conducted through the latter outward. The after-treatment does not materially differ from that of the radical operation. Healing takes place by covering the antrum and attic with epidermal epithelium. Both the attic and the antrum will now remain permanently open toward the auditory canal, a new membrane being formed only in exceptional cases.

The treatment, until complete healing occurs, will occupy from six to ten weeks.

Similar methods of operation have been proposed by Bondy and Heath.

OTOGENIC PARALYSIS OF THE FACIAL NERVE

Otogenic paralysis of the facial nerve develops in the course of an affection of the ear and the etiology of which can be connected directly or indirectly with the latter. Usually the paralysis is peripheral, ventral otogenic paralysis of the facial occurring only through endocranial otogenic affections. The paralysis is either complete or incomplete (paresis); there are also to be distinguished paralysis which sets in suddenly in the course of an auditory affection and one which runs a more chronic course.

Paralysis may further be divided into inflammatory and traumatic forms. The chief affections which are comprised in the former are those occurring in the course of acute or chronic inflammations—mostly suppurative—of the middle ear, furthermore those occurring in suppuration of the labyrinth and suppurative inflammation of the internal auditory duct (usually a part manifestation of purulent meningitis). Only very rarely is paralysis of the facial nerve an accompaniment of neuritis acustico-facialis. Lymphomatoses (leukæmia, pseudoleukæmia, chloroma, lymphosarcoma) may likewise be complicated by otogenic paralysis of the facial nerve.

Paralysis of the facial nerve occurring in the course of malignant tumors of the middle ear should be classed as inflammatory paralysis, owing to the suppuration of the middle ear with which it is usually associated. It may also occur in combination with acusticus tumor (especially in cases of glioma and angiosarcoma) by normal tissue being replaced by the tumor.

In the traumatic cases paralysis of the facial nerve occurs as a consequence of a traumatic injury of the auricular region or of a fracture of the base of the skull running through the petrous bone. Finally, a traumatic paralysis of the facial nerve may be caused by an operative injury of the nerve itself or its osseous canal.

Central paralysis of the facial nerve may occur in otitic abscesses of the temporal lobe. In cerebellar abscesses peculiar changes of the

facial nerve may occur if the abscess extends more or less centrally in the worm or if it exerts special pressure on the fossa rhomboidea or perforate into the fourth ventricle.

Anatomy.—The facial nerve turns from the crus cerebri outward and downward into the internal auditory canal, then runs over the acusticus laterally outward. It leaves the internal auditory duct at the foramen spurium canalis facialis and forms an angle of about 80° (lateral knee) backward and downward. It proceeds from here in the shape of an arch between the lateral semicircular canal and the vestibular window downward and backward, and leaves the petrous bone through the foramen stylomastoideum. In its course between the lateral knee and the vestibular window it crosses the nervus utriculo-ampullaris and the recessus utriculi.

There are no regular peripheral connections between the facial and auditory nerves. The bundle designated as nervus intermedius is situated in the internal auditory duct below the facial and on the vestibular nerve, sometimes running more closely to the former and sometimes to the latter, without, however, entering into any intimate connection with either, and fusing with the geniculate ganglion. The latter lies outside the internal auditory duct at the foramen spurium. In children under four years of age it lies more or less free under the dura; in older children and adults an osseous plate, belonging to the petrous bone, grows from behind over the ganglion. Only about two-thirds of the fibres of the facial nerve are interrupted in the geniculate ganglion; one-third turns posteriorly into the facial canal (canalis Fallopii) without suffering any interruption by the geniculate ganglion, but uniting at once with the fibrous part interrupted by the latter, and forming the round facial nerve bundle.

Where the facial nerve leaves the crus cerebri it contains marrow. The zone which contains marrow and the one which does not are sharply separated from each other, either in a straight or slightly convex surface. Gliotic, central, fibrous parts, protruding into the peripheral nerve, are but seldom to be found. The fibres composing the facial nerve are considerably thicker than those of the auditory. The cells of the geniculate ganglion are much larger than those of the vestibular or of the cochlear nerves. In the higher class of mammals there is a continuous connection between the geniculate ganglion and the upper—sometimes also the lower—vestibular ganglion. In other animals there are some varieties of continuous connections between the geniculate ganglion and the trigeminal ganglion. Rare clinical observations, especially on rheumatic affections in the regions of the trigeminal, facial, and auditory nerves, seem to indicate that similar connections occur exceptionally in man, but in my extensive anatomical material it has never been observed.

In its peripheral course the facial nerve sends out the following branches:

(1) *Nervus petrosus superficialis major.* This nerve represents an anastomosis between the second trigeminus branch (ganglion sphenopalatinum) and the facialis. It forms the white portion of the nervus Vidianus and runs through the semicanalis nervi Vidiani to the ganglion sphenopalatinum; it supplies the latter with motor fibres for the levator veli palati and with sensory fibres for the nasal mucosa. By the same route sensory fibres are supplied by the trigeminal ganglion for the facial nerve.

(2) *The nervus stapedius* branches off from the descending part of the facial canal and is the motor nerve of the stapedius muscle.

(3) *The chorda tympani* branches off below the stapedius nerve from the facial and arrives through the canalis chordæ to the tympanic cavity. It traverses the latter between the malleus and incus in an arch running forward and downward, and arrives through the fissura tympani squamosa between the two pterygoid muscles at the lingual nerve. The chorda contains secretory fibres at the bottom of the mouth as well as fibres for the perception of taste, the latter supplying the middle third of the tongue. A small branch is sent out by the chorda to the submaxillary ganglion and disappears in the sheath of the lingual nerve.

(4) *The nervus auricularis posterior* branches off below the stylomastoid foramen, and is the motor nerve for the posterior auricular muscle and the occipital muscle. At about the same level there is a branch for the stylohyoideus muscle and the posterior part of the digastric muscle.

(5) *The nervus anastomoticus* runs with the glossopharyngeus to the dorsal surface of the base of the tongue.

(6) *The plexus parotideus* is divided into 3 parts: the upper (rami temporales, rami zygomatici) runs over the zygomatic bridge to the temples and forehead, the anterior runs to the cheek, and the inferior crosses the lower loop of the masseter and runs along the submaxillary bone forward (nervus marginalis mandibulæ). The ramus of the facial nerve which supplies the platysma likewise branches off from the inferior plexus.

Pathological Anatomy.—The pathologico-anatomical picture of the peripheral otogenic paralysis of the facial nerve varies considerably. In most cases there are suppurative infiltrates along the nerve sheaths; besides there are inflammatory infiltrates in the latter and between the nerve bundles, especially in the area of the geniculate ganglion. In many cases the nerve is bathed in pus; in others the osseous facial canal is destroyed by a chronic ostitis of the middle ear or by trauma. After

destruction of the osseous wall, granulations or cholesteatomata may advance into the nerve and thus cause paralysis.

In paralysis due to hemorrhage there are extensive effusions of blood into the nerve itself, while extensive extravasations between the osseous canal and the nerve-sheaths do not impair the function of the facial nerve unless the blood penetrates into the nerve.

Trauma may injure the osseous facial canal, pulling at the nerve to a greater or smaller extent, or even tearing it.

In postoperative paralysis of the facial nerve there are extensive hemorrhages in the nerve itself, if the paralysis occurs immediately after the operation. The hemorrhages may also be secondary, due to the chisel, or there may be an operative injury of the osseous facial canal, of the nerve within, or it may be completely destroyed. In rare cases a postoperative paralysis may occur through splinters of the fractured facial canal being pressed against the nerve and leading to considerable compression of the same. Postoperative paralyses which gradually develop in from 3 to 6 days after the operation occur either by direct infection at the operation or indirect by acute neuritis of the facial.

Paralyses of the facial nerve which occur in the course of uncomplicated acute otitis are chiefly caused by congenital tearing of the osseous facial canal. Suppurative inflammatory infiltration of the middle-ear mucosa may be the immediate cause of an inflammatory paralysis of the nerve.

If the paralysis is caused by changes of the internal auditory duct, the bundles of the facial nerve have been destroyed by pus or granulations (complicated suppuration of the labyrinth). They may also be destroyed by tumors of the angle of the pons cerebelli or by pressure atrophy.

All forms of facial paralysis present the common anatomical picture of a more or less advanced varicose disintegration of the axis-cylinder and degeneration of the medullary sheaths.

Anatomical Course.—The peripheral fibres of the facial nerve have a high power of regeneration. The restoration of the fibres is favorably influenced by the curative process of the suppurative inflammation of the middle ear and the nerve-sheaths. Paralysis of the facial nerve due to hemorrhages is cured by resorption of the blood coagula. The regeneration occurs promptly if the local conditions permit, but even in cases of long standing the chances are favorable if the fibres are given an opportunity to recover, either by removal of any existing obstacles or by anastomosis with healthy motor fibres.

Clinical Symptoms and Clinical Course.—The most striking symptom of unilateral facial paralysis is the arrest of the mimic musculature on the affected side. Paralysis of the oral branch renders the angle of

the mouth immovable, the malar skin loses its normal tension, becomes flabby, and is either bulging outward or injected through the act of respiration. Paralysis of the frontal branch is followed by loss of the voluntary movements of the skin of the forehead. Paralysis of the sphincter of the lid causes rather considerable lagophthalmos, accompanied at first by increased, later by decreased lachrymation. In some cases, especially in children, lachrymation is entirely arrested, and the bulbar surface becomes dry. The defective or totally suppressed ability to close the lid exposes the bulb to the possibility of traumatic injury; during sleep there is danger of contracting keratitis from drying up of the corneal moisture. Iridocyclitis or panophthalmia may develop in rare cases. If paralysis has existed for a long time, exophthalmos may result. Congenital paralysis of the facial nerve, occurring in early infancy, leads to asymmetry of the skull, the affected side growing larger than the healthy one.

Localization.—If all the branches of the parotid plexus are involved in a peripheral otogenic paralysis of the facial nerve, the seat of the lesion lies in the trunk of the nerves centrally from the parotid plexus.

If the nerve has been injured above the branching off of the auriculotemporal nerve and below the chorda, there is, aside from paralysis of the mimic musculature, loss of motility of the neck and head. If the chorda tympani is involved, there will be disturbances of taste in the tongue; besides, there is a sensation of burning of the tongue on the affected side in the first stages of the attack. When the chorda has been destroyed, the potassium test of the saliva will be negative. Paralysis of the stapedius nerve causes slight reduction of the hearing acuity and subjective noises. If the seat of the paralysis is in the internal auditory duct, there will be functional disturbances in the area of the superficial petrous nerve. Unilateral paralysis of the velum palati occurs only in rare cases.

In central paralysis of the facial nerve there is never a complete unilateral paralysis of the peripheral area of the nerve; there is always partial paralysis, affecting either the upper or lower area of the facial nerve, these regions being separated by the zygomatico-oculo-nasal line.

Complete unilateral paralysis of the facial nerve gives rise to many disturbances. Mastication and drinking are difficult, as the food tends to escape toward the affected side, and at first patients are unable to prevent liquids from running out of the affected side of the mouth or solid food from protruding. After a while, however, they acquire a special knack enabling them to eat; with the aid of their fingers they manage to push solid food toward the healthy side, while liquids may be controlled by inclining the head toward that side. Whistling and sucking are impossible at first. In facial paralysis occurring in the course

of chronic middle-ear suppuration, the chorda and stapedius symptoms are of rare clinical occurrence. Paralysis of the frontal branch does not give rise to serious complaints.

Unlike rheumatic paralysis, inflammatory otogenic paralysis of the facial nerve occurs but rarely as an apoplectiform affection. As a rule, the otitic paralysis sets in with slight disturbances in the region of the facial nerve (involuntary twitching, periodical lesions of the oral and ocular branches), and it is only after some days or weeks that the affection reaches its full extent.

Diagnosis.—In the majority of cases the otogenic character of facial paralysis can be easily recognized from the history. This is especially true of those cases which develop in the course of an acute or chronic suppuration of the middle ear. Where, however, this affection is not present and the tympanic membrane is intact, the differential diagnosis from rheumatic paralysis may cause difficulties. The guiding points lie in the history and the knowledge on the part of the patient whether the paralysis has slowly developed to its full extent in the course of several days or whether it was from the first of an apoplectiform nature. In the former case it points to otogenic, in the latter to rheumatic paralysis.

Central paralysis attacks either the upper or lower fractures of the facial nerve, so that there is never complete unilateral paralysis. Furthermore, in every form of peripheral paralysis of the facial nerve the electric excitability of the peripheral nerve is pathologically changed; in central paralysis it is normal.

Prognosis.—The extent of the paralysis is of no particular consequence for the prognosis. Generally speaking, incomplete paralysis may be said to have a better chance than a complete one, but often enough there are exceptions. Of great importance is the rate at which the affection has developed. Thus, postoperative paralysis, which is complete from the first, is prognostically unfavorable, while cases which have gradually developed (not traumatic) are the more favorable the longer it has taken for the affection to develop.

In peripheral paralysis a more accurate prognosis can be made by an electrical examination. Patients with faradic excitability have a chance of rapid and complete cure (three to six weeks). This holds good for all acute and most chronic cases of paralysis of the facial nerve. Chronic cases which can not be cured in spite of preserved faradic excitability are very rare. Paralysis with destroyed faradic excitability are to be regarded as serious cases, regardless of whether the paralysis is complete or incomplete.

However, these cases have still a chance of recovery if the reaction of degeneration can be produced and the galvanic excitability increases

during two or three observations. In that case the reaction of degeneration can be produced with a constantly decreasing current; finally it disappears altogether and the normal galvanic and later the faradic excitability will be restored.

Cases in which the galvanic excitability of the nerve is still preserved also give a favorable prognosis, as after a short time the faradic excitability is usually restored.

The prognosis is unfavorable if the reaction of degeneration is combined with gradually diminishing excitability of the muscle; in these cases the production of the reaction of degeneration requires a constantly increasing strength of current.

Similarly the prognosis is unfavorable in all cases in which the reaction of degeneration exists in mere traces, and the galvanic excitability is destroyed with the exception of rudimentary remnants, especially at the angle of the mouth and the eyelid.

Course.—It is difficult to form an opinion as to the length of time required to effect a complete cure. In prognostically favorable cases of serious paralysis with destroyed faradic excitability the time required is from three to six months, but the motor function on the affected side may nevertheless remain weak for a considerable time, perhaps for years; in other cases the function of the nerve will never be completely restored.

But even in cases in which no complete cure is possible a certain balance is restored, and with the aid of the trigeminus of the affected side or the facial and trigeminal nerves of the healthy side, and by practice, the most serious disturbances in the ingestion of food and speech will disappear.

The cases which defy cure may be divided into two groups,—paralysis which persists in the flabby form, which occasionally leads to atrophy of the facial skin and of the malar adipose tissue; and paralysis in which there is gradual development of muscular contracture. Moderate contractures are favorable for the cosmetic effect. The normal facial contour is restored and the closure of the lid improves. But if the contractures continue to increase, the disturbance will assume considerable proportions, the angle of the mouth will be distorted and there will be passive permanent closure of the lid.

Treatment.—Should facial paralysis be caused by chronic suppuration of the middle ear, immediate radical operation is indicated. In the majority of these cases facial paralysis is a very bad sign, showing as it does that, owing to the spreading of suppurative ostitis or a gradually growing or acute suppurative cholesteatoma, the labyrinth and endocranium are likewise endangered. It is necessary, therefore, in each case of facial paralysis occurring in the course of chronic suppura-

tion of the middle ear, to examine the patient very carefully for labyrinthitis or an endocranial otitic affection.

Light cases of facial paralysis (with preserved faradic excitability), occurring in the course of acute middle-ear suppuration, admit of conservative treatment of the latter, provided it runs a perfectly normal course, which means normal temperature, no pain, no mastoid symptoms, no descent of the posterosuperior wall of the auditory duct, and gradual decrease of the suppuration with increasing hearing acuity. In all other cases immediate antrotomy is indicated.

If the paralysis is postoperative, the bandage should be immediately and completely removed, so that a search for bone splinters may be instituted and a reliable drainage for the middle-ear spaces provided for. The retro-auricular wound should be kept open.

Where the paralysis is a part-manifestation of suppuration of the labyrinth, the latter should be resected, and, should there be simultaneous signs of suppurative meningitis, the internal auditory duct should be exposed.

Favorable results have been reported of the operation of opening the facial canal and exposing the facial nerve in the middle ear in cases where postoperative paralysis has been present for a long time. After traumatic paralysis due to injury or complete severing of the nerve and its osseous canal, the operation of broadly opening the facial canal may likewise be attended with success, provided it is possible to remove all obstacles which have prevented reunion of the divided nerve-ends, or to chisel out an osseous groove serving as a kind of splint for the severed nerve-ends.

As to the rest of the conservative and surgical treatment of an otogenic paralysis of the facial nerve, the same therapeutic rules hold good which are followed in facial paralysis generally. In light cases faradic treatment, applied for 5–10 minutes every day, is sufficient. The highest electric energy is applied which can be borne by the patient without discomfort.

Should the faradic excitability be destroyed, the galvanic current is resorted to. With excitability preserved from the nerve, the best plan is to apply one electrode to the trunk of the nerve below the lobulus, and the other to an indifferent place, such as the hand, chest, or neck. Besides, galvanization of the paralzyed muscle itself should be applied.

With the excitability from the nerve destroyed, the treatment must be confined to the paralyzed muscles.

The electric treatment is considerably aided by light massage of the facial muscles and mimic exercises carried out by the patient before the looking-glass. Hot-air treatment, perspiration, and laxatives are

only of value in otogenic paralysis of the facial nerve if the affection has developed in the course of simple acute otitis media.

Old cases of otogenic facial paralysis belong to the domain of the surgeon, who will endeavor either to cure the paralysis by anastomosing the facial nerve with the accessory or the hypoglossus nerves; or he must content himself with overcoming the most disfiguring or objectionable disturbances by plastic operation.

In anastomosis the following methods are to be distinguished :

(1) Anastomosis between the facial nerve and the motor branch of the accessory nerve. A skin incision 4–5 mm. long and slightly curved backward runs from the insertion of the concha to the posterior edge of the sternocleidomastoid muscle. The soft parts are pushed aside with a blunt instrument until the pedicular process can be felt. Immediately behind the latter there is the trunk of the facial nerve, enclosed in a thick sheath. This is dissected free and pulled forward with a small forceps. In doing so, it is useful to roll up the nerve-trunk upon the forceps, so that the nerve may be intersected as high as possible in the facial canal. The end of the facial nerve is caught by a silk suture and cut off in an oblique direction. Now the accessory nerve is exposed and followed upward to the bundle intended for the deltoid muscle. At this place an end-to-end anastomosis is usually done by cutting the deltoid branch of the accessory nerve in an oblique direction and uniting the cut central end with the peripheral end of the facial nerve by two interrupted silk sutures. The use of silk sutures in nerve anastomoses has been recommended because an inflammatory reaction is advantageous for the rapid union of the anastomosed nerves.

If lateral anastomosis is preferred, the same motor branch of the accessory nerve is nipped with the scissors and incised. The neurilemma being thus removed and a number of severed nerve-fibres exposed, the end of the facial nerve is implanted and fastened with interrupted sutures. A thin drainage strip (isoform or iodoform wick) is inserted from the lower traumatic angle to the anastomosis, and the skin wound is closed by Michel's clamps.

(2) Anastomosis between the facial nerve and the hypoglossus nerve. The skin incision runs from the lower insertion of the concha straight to the hyoid bone for about 4 cm. The trunk of the facial nerve is dissected free as in anastomosis with the spinal accessory nerve. The hypoglossus nerve is next searched for in the trigonum digastricum or before the digastricus, after which a lateral anastomosis can be done by a superficial incision of the hypoglossus, or an end-to-end anastomosis by intersecting the hypoglossus. A drain is conducted through the anterior traumatic angle to the anastomosis, and the skin wound closed by Michel's clamps.

Indications for anastomosis are in fresh cases of facial paralysis where the traumatic changes of the facial canal and nerve render conservative treatment absolutely hopeless, as for instance in compound fractures, in which not only the nerve has been severed, but part of it has been destroyed, and the osseous facial canal has undergone serious injury. Anastomosis is also indicated in inflammatory paralysis when part of the nerve and osseous canal has been completely destroyed by chronic suppuration of the middle ear or cholesteatoma.

In cases where the continuity of the facial nerve has not been impaired, anastomosis should not be considered until all conservative measures have been found unavailing. It is then important not to miss the proper time for surgical interference. In complete paralysis of the facial nerve, with destroyed faradic and diminishing galvanic excitability from the muscle and negative excitability from the nerve, anastomosis is usually indicated if after 6–12 months' conservative treatment there are no favorable changes in the excitability and no functional improvements. In cases that have been paralyzed for more than two years there is usually no more chance of benefiting the patient by anastomosis, because during that length of time the entire nerve is usually so degenerated that a nutritive union with a healthy motor nerve will be impossible.

Postoperative Course.—Facial anastomosis being only resorted to in complete peripheral paralysis, resection of the paralyzed facial nerve will at first not give rise to external improvement. It has also been uniformly observed that small remnants of retained function in the regions of the eyelids and the angle of the mouth will not be impaired by central resection of the facial trunk. This fact goes to prove that these remnants of innervation are not referable to the paralyzed nerve; they are the result of vicarious function of ramuli of the motor trigeminus of the same side or of the facial nerve of the healthy side.

Successful end-to-end anastomosis between the facial and accessory nerves leads almost always to paralysis of the shoulder on the operated side; that between the facial and hypoglossus to unilateral paralysis of the tongue. These manifestations will often completely disappear in from eight to twelve months; in other cases they will permanently continue. In lateral anastomosis the region of the accessory and hypoglossus may not be functionally disturbed; in most cases, however, paralysis will occur and disappear after four to twelve months.

Successful treatment depends upon various circumstances. Thus, the degeneration of the facial nerve must not be excessive and, as has been shown by experimental studies, the remaining medullary sheaths must be of sufficient length and in fairly normal condition.

The first symptom of improvement in favorable cases occurs in

from six to twelve months, shorter periods being rare. These cases require regular galvanic treatment, in the course of which the patient may report improvement in mastication and drinking, later in whistling, and finally in aspiration.

A perfectly ideal success with restoration of the entire mimic musculature, however, is of rare occurrence. In the cured cases independent innervation of the facial nerve is impossible. The voluntary use of the mimic musculature is combined with movements of the shoulder in anastomosis between the facial and accessory nerves, or by movements of the tongue in anastomosis between the facial and hypoglossus nerves. Occasionally voluntary contractions of the shoulder and lingual muscles are accompanied by passive twitching in the region of the facial nerve. It is questionable whether these patients will ever reacquire the ability of innervating the facial singly. This would require regeneration and isolation of the cortical motor fields of the facial nerve, and it is assumed that this would more easily take place after anastomosis between the facial and hypoglossus nerves, because the cortical field of the latter closely approaches the lower cortical field of the former. On the other hand, the facial nerve is at a much greater distance from that of the accessory nerve, so that permanent associated movements of the shoulder have to be expected.

Gersuny proposed a plastic method for the cure of flaccid paralysis where the soft parts of the cheeks and malar skin have been considerably stretched. He conducts a silk or silver wire suture from the zygomatic region to the angle of the mouth and back to near its starting-point, in order to raise the angle of the mouth by contractures. The suture ends are knotted over a small iodoform roll. The suture is allowed to remain *in situ* from four to six weeks, and the result is said to be favorable in producing medium-grade contractures.

In chronic flaccid paralysis or high-grade contractures in the region of the eyelid, a lid plastic operation is to be considered. For the details of the various methods, text-books on ophthalmology should be consulted. It has already been mentioned that high-grade muscular contractures represent the worst result of a permanent facial paralysis, and the best way to prevent their development is regular galvanization from the muscle for ten minutes once or twice weekly and continued, if necessary, for several years; and in addition massage, hot-air baths, and inducement of perspiration.

Many patients learn to massage their face so well that the muscles retain their tone. Usually a pinching of muscles followed by a thorough rubbing upward toward the eye is sufficient to mantain some degree of action.

X. AFFECTIONS OF THE CAPSULE OF THE LABYRINTH

I. EXOSTOSES OF THE LATERAL WALL AND OBLITERATION OF THE WINDOW OF THE LABYRINTH

Exostoses are seldom observed at the lateral wall of the labyrinth. They occur occasionally in chronic suppuration of the middle ear at the corners of the fenestræ, at the auricular ossicles, at the promontory, or at the eminence of the lateral semicircular canal. Leidler has called attention to diffuse new-formation of bone at the walls of the tympanic cavity in cases of acquired atresia of the auditory duct.

According to past experience it is not impossible that even a fracture or pathologic fistula of the labyrinth may heal. Congenital exostosis at the capsule of the labyrinth is exceedingly rare; it appears only as part manifestation of a pronounced general deformity of the body with severe malformation of the ear (anencephaly, synotia).

Obliteration of the windows of the labyrinth may be due to connective tissue or bone formation. Obliteration by connective tissue is the result of catarrhal adhesive processes of the middle ear or of a middle-ear suppuration. Both window corners are replete with cicatricial tissue, while coarse connective-tissue layers extend to the stapes plate and the membrane of the cochlear window. Window corners filled with connective and fatty tissue have been observed in cretins.

The mucous tissue which is normally found in the tympanic cavity of the new-born will persist longer than usual in rhachitic children and completely fill up the window corners.

Ulceration of the mucous membrane and superficial osseous erosions at the middle wall of the tympanic cavity, and osseous atresia of both labyrinth windows, occurring in chronic suppuration of the middle ear, seldom can be cured. The corner of the cochlear window is completely filled with bone, and the entire stapes may be replaced by osseous proliferation from the vestibular window.

Obliteration of the labyrinth windows (stapes ankylosis and occlusion of the cochlear window) may also occur in otosclerosis if the pathological bony proliferation of the lateral wall of the labyrinth has involved the labyrinth windows.

Exostoses of the lateral wall of the labyrinth, without obliteration of the cochlear windows, do not cause any particular cranial symptoms. Considerable reduction or complete destruction of the hearing acuity of the affected ear in the presence of exostosis is due to the fact that those affections in which exostoses occur lead of themselves to consider-

able injury or destruction of the membranous labyrinth, of the nerve-end places, and of the eighth nerve.

Diagnosis.—In exposed middle-ear spaces—*i.e.*, after destruction of the attic or antrum wall—exostoses may sometimes be otoscopically demonstrated; generally, however, they are accidentally met with on the occasion of a radical operation. Obliteration of the labyrinth windows is indicated by the negative result of Gellé's test.

Treatment.—There can be no question of any treatment except in obliteration of the window corners by connective tissue in the presence of chronic adhesive processes. Politzer recommends resection of the ligament and exposure of the window corners. Wherever the membranous cochlea and Corti's organ have been preserved, or at least not materially injured, it is possible to obtain considerable and permanent improvement of the hearing acuity by this operation. The indication for it must be given by the test with the c4 tuning fork, establishing the upper sound limit. If the perception of the c4 fork for air-conduction is only slightly shortened (2–10 seconds), resection of the cicatrices will surely lead to a favorable result; if, however, the perception for c4 is considerably reduced and the high-sound limit quantitatively impaired, endotympanic surgery offers no chances of success.

II. ACUTE, SUPPURATIVE PARALABYRINTHITIS

Suppurative paralabyrinthitis is characterized by the suppurative softening and absorption of the bone in the area extending from the osseous capsule nearly to the labyrinth spaces. It may become more extensive in the angle of the semicircular canal. The superficial bone erosions of the middle wall of the tympanic cavity likewise belong to the group of affections designated as paralabyrinthitis.

Acute suppurative labyrinthitis usually presents no symptoms either at the beginning or in its course. The diagnosis can clinically be made in rare cases from the expulsion of the smallest sequestra from the paralabyrinth, or from the occasional recognition at a mastoid operation. The affection is relatively frequent in childhood and is observed oftener in the case of acute than of chronic middle-ear suppuration.

In cases of acute middle-ear suppuration the cause of paralabyrinthitis is usually a mastoid suppuration with manifestations of retention. The danger of paralabyrinthitis is especially great in early age as long as the petrous bone possesses large diploic spaces filled with marrow, or pneumatic spaces. In the chronic cases paralabyrinthitis is nearly always caused by middle-ear suppuration, which may be either tuberculous or complicated by cholesteatoma.

The treatment consists in the radical operation and surgical removal of the affected bone with the sharp spoon; operative injury of the labyrinth must, of course, be avoided.

The prognosis is not unfavorable in acute uncomplicated cases, but it is advisable to keep the retro-auricular aperture open as long as possible in order to prevent the possibility of a weak superficial healing, recesses, or trabeculæ.

In the chronic cases there is danger of postoperative serous labyrinthitis. If paralabyrinthitis is caused by a tuberculous middle-ear suppuration, there is a possibility of an acute tuberculous suppuration of the labyrinth and the danger of tuberculous meningitis.

III. FISTULÆ OF THE LABYRINTH

A fistula of the labyrinth is an abnormal communication of the labyrinth spaces with neighboring spaces caused by a pathological process. From a topographical point of view, these fistulæ have, therefore, to be divided into intra- and extracranial, and among the latter the tympanic variety occupies a distinct place. Intracranial fistulæ of the labyrinth lead into the middle or posterior cranial fossa, the extracranial ones into the middle-ear spaces (tympanic cavity and antrum) or into the fossa jugularis. Fistulæ of the endolabyrinth may be classed as a special group, but they are usually congenital defects. They occur from congenital arrest of development, preventing the formation of the normal bony septa in the embryonal labyrinth. In the most pronounced cases the labyrinth is a single space with hardly any divisions, while in cases of less pronounced malformation there are abnormal connections between the spaces of the labyrinth. Even the septum between the latter and the posterior semicircular canal may be absent, which is especially observed in congenital deafness and congenital absence of the scala of the cochlea.

Acquired fistulæ may occur either through preformed or pathologically developed routes. The preformed routes are the fenestra vestibuli, fenestra cochleæ, aquæductus vestibuli, aquæductus cochleæ, and internal auditory canal. In these cases a fistula occurs by the destruction of the corresponding bones or soft parts through a pathological process.

Fistulæ occurring over routes not anatomically preformed may of course start from any point in the labyrinth, but also from any region of the endocranium or the ear. There are, however, certain places of predilection in the labyrinth,—namely, where there is only a relatively thin bone protection against the endocranium, the middle-ear spaces, or the fossa jugularis. Such places are the following: (1) promontory; (2) the lateral crus of the external semicircular canal; (3) the vortex of the upper semicircular canal or, in children up to the fourth year, the entire upper semicircular canal; (4) the sinus end of the sagittal semicircular canal; (5) in infancy, the commissure of the semicircular canal.

It may be mentioned that, even in normal conditions, the dimensions of the bony layer at the places mentioned are very variable, and

that infantile affections of the osseous system, notably rhachitis, may be the cause of these superficially situated parts retaining their original topography far beyond the infantile age.

Fistulæ of the labyrinth are divided according to their origin into excentric and concentric varieties. An excentric fistula is one which originates by a suppurative process within the labyrinth and breaks through the capsule and the petrous bone. The concentric fistula occurs from an affection spreading from the surrounding parts to the capsule of the labyrinth, leading to a fistulous perforation of the labyrinth from without. It is clear, therefore, that excentric fistulæ show the result of a suppuration in the labyrinth, and the concentric ones show the beginning of a suppuration in the labyrinth.

The endocranial fistulæ are subdivided into intradural and extradural ones; the extracranial fistulæ into subperiosteal and intravenous (perforating into the bulbus of the vena jugularis or into the sinus sigmoideus).

Among the tympanic fistulæ of the labyrinth we distinguish between submucous and perforating. The former occur through the fistulous opening being closed against the middle-ear spaces owing to the pathologically thickened tympanic mucosa, and the latter through the fistula discharging its contents against the middle-ear spaces.

Furthermore, fistulæ may have different relations to the labyrinth spaces, which give rise to the following distinctions: paralymphatic, perilymphatic, and endolymphatic. The paralymphatic fistulæ occur through the destruction of the osseous capsule, the perilymphatic connective-tissue capsule remaining intact; in the perilymphatic fistulæ a communication has been established between the perilymphatic spaces and the fistular canal, and the endolymphatic fistulæ communicate with the endolymphatic spaces.

A study of the various groups with regard to the last-mentioned point will result in the following: The excentric fistulæ are absolutely endo- and perilymphatic. The excentric endocranial fistulæ are intradural if they have developed by way of the cochlear aqueduct or of the internal auditory canal; they are extradural if developed via (1) the vestibular aqueduct; (2) the frontal semicircular canal; (3) the sagittal semicircular canal; (4) the commissure of the semicircular canal; (5) the ductus and sulcus endolymphaticus. These fistulæ can only become intradural after destruction and fistulous perforation of the dura.

As to the concentrically originated fistulæ, it can be stated that, if the affection is slight or takes a very slow course, the changes will be arrested at the external surface of the perilymphatic capsule of the labyrinth. The reason for this may be either that after destruction of the bone the pathological process has no tendency to spread any further,

or that during the long period required for destroying the osseous wall reactive changes have taken place in the labyrinth spaces by new-formation of connective tissue or bone, which prevent the fistula from continuing its course into the peri- or endolymphatic spaces.

Submucous tympanic fistulæ are especially found after excentric destruction of the cochlear window. The corner of the latter has usually been closed for some time against the tympanic cavity by connective-tissue layers. The fistular contents (pus) now exude into the corner of the round window, but cannot reach the free tympanic cavity.

Symptoms and Course.—The formation of labyrinth fistulæ usually occurs under violent irritative manifestations of the labyrinth. The permeability of the fistula in the area of the static labyrinth is indicated by a violent attack of labyrinthine vertigo. In excentric fistulæ these attacks of vertigo have already been preceded by a few others of minor intensity, while in concentric fistulæ the patient never had an attack before, but the violent attack is followed later by a series of long-lasting or repeated lighter attacks. The auditory acuity undergoes sudden deterioration, and in some cases instant deafness will set in. The permeability of excentric fistulæ, however, may occur without giving rise to any symptoms if the membranous labyrinth had already been destroyed for some time previous to the development of the fistula. For labyrinth fistulæ in suppurative paralabyrinthitis, see p. 271.

The formation of a labyrinth fistula may be followed by diffusely spreading destruction of the osseous capsule which may cause the destruction of the lateral wall of the labyrinth; in isolated cases the entire labyrinth may be sequestered. This end result occurs especially in excentric fistulæ which are only part manifestations of a chronic, diffuse, complicated suppuration of the labyrinth. In concentric fistulæ the chances of a cure are favorable, provided a middle-ear operation has been done in time, consisting in antrotomy for acute or subacute, and in the radical operation for the chronic cases. Closing of the fistula will take place by connective-tissue scars or new bone formation.

The diagnosis is principally based upon the history concerning the first attack of vertigo or the clinical observation of such an attack. In typical cases the occurrence of vertigo is contemporaneous with the permeability of the fistula; concentrical fistulæ especially are accompanied at the time of their breaking through by typical and sometimes stormy symptoms of vertigo and equilibrial disturbances of the labyrinth. Acute diminution of the auditory acuity or sudden deafness is not a rare occurrence. In fresh cases the static labyrinth usually shows pathologically increased (prolonged) reflex excitability (Stage II of inflammatory irritation, see p. 269). For clinical fistular symptoms, see p. 93.

Treatment.—Fistulæ occurring in acute suppurative paralabyrinthitis will heal spontaneously after antrotomy or the radical operation has been performed. If the fistula is due to acute or subacute middle-ear suppuration, antrotomy is indicated; if due to chronic middle-ear suppuration, the radical operation should be done. After the operation the irritative phenomena rapidly disappear, and, as a rule, no more vertigo will occur as soon as the retention of pus in the middle ear and the consequent increase of pressure upon the fistula have been removed. In cases where a concentric fistula has been caused by an acute or subacute middle-ear suppuration, the hearing acuity will be improved or restored to normal. The hearing acuity in cured chronic cases is rarely satisfactory; in fact, deafness may gradually set in a long time after the fistula has been closed. This contingency is attributable to degenerative atrophy of Corti's organ.

IV. OTOSCLEROSIS

The etiology of otosclerosis is not known. The suggestion of Habermann and others that it is a metaluetic or hereditary syphilitic affection has been refuted by Arzt, O. Beck, and others.

As it is just in hereditary syphilis that Wassermann's reaction is very reliable, the test in otosclerosis should be positive in a large number of cases. Such, however, is not the case by any means; on the contrary, it has long ago been demonstrated that otosclerosis is the type of hereditary affections of the ear.

Genealogical statistics have proved the continuous heredity of otosclerosis, even in the branch lines of affected individuals, and sometimes omitting an entire generation.

Otosclerosis may also be a part manifestation of hereditary degenerative changes of the central nervous system. Thus, there are forms of psychic affections and otosclerosis in descendants of the same family. In other cases a tendency to heredity is associated with otosclerosis. Hammerschlag is right, therefore, in describing the affection as one of hereditary difficulty of hearing.

Anatomy.—Politzer recognized otosclerosis as an affection of the osseous capsule of the labyrinth. When examining the petrous bone in otosclerosis, there are found pathological foci of bones which, in the shape of sharply demarcated tumors, often replace the normal bone of the capsule. Sometimes the pathologically changed bone is not clearly separated from the normal one, or both may gradually fuse into each other. The pathological foci are composed of bone that abounds in cells and lime. The histological examination shows that petrous bones which are normally already decalcified still contain lime in the pathologically changed portions. In the histological examination, the normal bone

stains bright red under application of hæmatoxylin-eosin, while the otosclerotic foci assume a reddish-blue or deep-blue color. As compared to the compacta of the normal petrous bone, the otosclerotic foci have very large hollow spaces which are freely permeated by pathologically enlarged blood-vessels. There is a rather dense net-work of connective tissue spread out between the bone and the vessel walls. The favorite seat of these foci is the lateral walls of the labyrinth. As they become gradually enlarged, the osseous foci lead to gradual thickening of the lateral wall of the labyrinth, constriction and finally osseous obliteration of the window corners, and ankylosis of the stapes.

In some cases the osseous foci protrude, tumor-like, into the labyrinth spaces (vestibular cistern, scala tympani). They occur less often in the area of the semicircular canals, body of the cochlea, or near the internal auditory canal. The topographical relation of the pathological osseous foci to the surface of the labyrinth is of importance. It may be assumed that otosclerotic foci may exist without symptoms, as long as they have not reached the windows of the labyrinth and the inner surface of the capsule. Findings in congenitally deaf individuals and cretins justify the assumption that the otosclerotic osseous foci are chiefly of congenital origin, situated in infants and young children in the petrous bone itself, as are the cartilaginous interglobular spaces; thus, they reach at that time neither the inner nor outer surface of the capsule nor the windows of the labyrinth. It is not before the period of puberty that increased growth of the pathological osseous foci seems to occur. They now extend to the windows of the labyrinth and reach the labyrinth spaces or the dural lining of the internal auditory canal. This assumption is supported by the clinical fact that the first important clinical symptoms in a great number of cases of a pronounced hereditary character do not occur until the period of puberty. Both ears are mostly attacked, but not in the same measure nor at the same time. Restriction to one ear is a rare occurrence.

According to their anatomical structure, the pathological osseous foci of the capsule have nothing to do in an anatomical sense with the ordinary sclerosis of the bones. The name of "otosclerosis" is, therefore, not justified, as it only designates the clinical conception of the affection. Attempts have, therefore, been made to coin a correct expression for the anatomical changes. Siebenmann's "spongiosis of the labyrinth capsule" must be rejected, as the pathological osseous foci are totally different from normal spongiosa of the bone. The hollow foci of spongiose bones are filled with medullary tissue; those in otosclerosis are filled with pathologically enlarged and new-formed blood-vessels. The expression "stapes ankylosis" describes only part of the cases correctly, as in many cases no such ankylosis occurs and the bony changes

are limited to other places of the capsule or to regions of the cochlear window. I believe I have found a correct term in "osteitis vasculosa." Indeed, the impression gained from the histological picture is that the otosclerotic foci are the result of a non-suppurative inflammation of the bones, which has led to the pathological enlargement of the blood-vessels, to increased cell-formation, and to increased lime deposits in the bone.

The idea of otosclerotic foci being the end result of a periostitis of the capsule is only borne out by a few specimens in my collection, whereas in many cases of typical otosclerosis any periosteal changes cannot be seen.

The anatomical changes of the capsule itself are sometimes associated with changes of the tympanic and the membranous labyrinth. The changes of the tympanic cavity are characterized by diffuse thickening of the mucous membrane of the middle tympanic wall and by considerable osmosis, increased vascularization, and hyperæmia of that mucosa. Serous osmosis of the mucosa of the middle ear may also lead to accumulation of a serous exudate in the tympanic cavity. As matter of fact, cases of otosclerosis have been observed which set in under the clinical manifestations of an exudative middle-ear catarrh. Such exudates, however, are not demonstrable in older cases.

The changes of the internal ear consist in degenerative atrophy of the sensory epithelium and the eighth nerve. In some cases these destructive changes involve the entire labyrinth; in others the vestibularis is spared. There is a great difference in the chronology of the capsular changes and those of the membranous labyrinth as compared to the auditory nerve. According to the clinical course, the rule will hold good in most cases that the degeneration of the sensory epithelium and auditory nerve develops secondarily upon advanced changes of the capsule. In other cases the changes of the capsule and membranous labyrinth appear to set in synchronously. Kalenda's investigations have shown that the symptoms furnished by the degeneration of the labyrinth may occur previous to the clinical symptoms of otosclerosis in cases which according to their later course or to the functional findings have to be diagnosed as otosclerosis.

Symptoms.—The most striking early symptom of otosclerosis consists in subjective ear noises of different kinds, such as the roaring of water, buzzing, humming, whistling, pulsating knocks, etc. They are increased by rush of blood to the head following physical exertion, excitement, and all efforts which increase the blood-pressure. In many cases the subjective noises are so violent that they molest the patient more than his impaired hearing. In other cases again they are less obtrusive or completely absent. The degree to which hearing is impaired varies considerably and depends in the first place on the stage of the affection. It is intelligible that hearing should be less impaired in the

beginning than at later stages. Generally speaking, gradually increasing difficulty of hearing is characteristic for otosclerosis. In some cases the hearing acuity decreases rapidly, so that it requires but a few years for a high degree or total deafness to establish itself. In other cases the progressive character of the affection is less pronounced, so that even after 20 or 30 years the hearing ability is still considerable. Even temporary improvement in the hearing acuity has been observed in rare cases. Physical exhaustion, mental excitement, psychic impressions, disturbance of the general health, may considerably impair the hearing, although at first only temporarily. Affections of the nasopharyngeal tract likewise exert an influence upon the degree of hearing ability.

If the static labyrinth is involved, there will be vertigo and equilibrial disturbances. The paroxysms of vertigo are usually not severe, and the equilibrial disturbances are restricted to the duration of the vertigo. A later symptom of otosclerosis consists in violent pains in the auricular region which are sometimes accompanied by reactive hyperæmia. There are also changes in the voice. When the hearing acuity is considerably reduced and the labyrinth is still intact, the patient perceives his own voice greatly intensified and even roaring. He endeavors to correct this by adapting a very much softer key. In considerable impairment of the auditory acuity, speech becomes drawling and the articulation indistinct. This is especially the case if hearing has already been impaired at juvenile age.

A frequent symptom is *paracusis Willisii,* which consists in improved hearing amid great noise, such as carriage or railway riding or loud music. Politzer explains this by assuming mobilization of auricular ossicles, the articulations of which had become rigid and now become more capable of conducting sound. At the same time the equilibrium of the auditory-nerve termination is disturbed, which facilitates the perception of sound. According to Urbantschitsch, this phenomenon is due to increased excitability of the auditory nerve caused by the concussion. Occurrence of hyperæsthesia in otosclerosis may be followed by grave general nervous manifestations.

Fröschels states that the sensation of titillation in the external auditory meatus is often reduced or absent.

Course and Prognosis.—The course of otosclerosis is unfavorable, but the various cases differ considerably in regard to prognosis. In a general way it may be said that the prognosis is the less favorable the earlier in life the affection has set in. The most unfavorable cases are those in which the impairment of hearing dates back to childhood. The first important deterioration is to be apprehended at puberty. Then there is rapid further diminution, so that at the age of about 30 or 40 there may be considerable loss of hearing ability.

The *prognosis* is more favorable where the first signs of impaired hearing occur between the 20th and 30th year. In these cases the pathological process may be arrested for a time, and there may even be transitory improvement.

Otosclerosis is often associated with secondary psychic manifestations. Patients appear apprehensive and depressed, this impression being increased by the pathological changes of the voice which will appear at a later stage. Children suffering from otosclerosis can follow their lessons only with the greatest exertion and display of the closest attention. For this reason general neurasthenic complaints will develop early, together with a tendency to pathological introspection. Patients are apt to regard all kinds of extraneous matters as the cause of the occurrence or exacerbation of their trouble, and consequently develop early in youth a tendency to solitude and seclusion. This is intelligible when it is considered that school education is imparted to the growing child chiefly by word of mouth and that progressively deaf children have therefore enormous difficulties to overcome. As soon as the hearing acuity has diminished to 10–13 feet, the child is unable to follow his lessons any longer, and he should be instructed separately or assigned to a class for those difficult of hearing. The uncertainty of understanding strangers may also lead to numerous misunderstandings, psychic conflicts, and despondency.

Diagnosis.—Politzer deserves the merit of having correctly formulated the clinical conception of otosclerosis. Accordingly, the diagnosis of typical otosclerosis is based on the following findings: The tympanic membrane and Eustachian tube are normal. Ventilation of the middle-ear spaces is perfectly normal. Functional test shows in the typical forms an obstacle to conduction (pathologically elongated bone-conduction, c4 normal or slightly shortened, upper sound-limit normal). In atypical otosclerosis, labyrinth symptoms may likewise be present (lowering of the upper sound-limit, in advanced cases sound-islands). The affection shows a distinct hereditary and progressive character and is associated with subjective hearing perceptions. In simultaneous involvement of the static labyrinth there are vertigo and equilibrial disturbances. As to transitory forms, see p. 236.

Early diagnosis in childhood may present difficulties owing to the fact that the condition of the tympanic membrane sometimes simulates a secretory catarrh. In these cases the diagnosis is often made on the proof of the hereditary character of the affection, and often also on the fact that after removal of the exudate insufflation of air will produce but slight improvement in hearing, if any. In ordinary exudative catarrh, however, the hearing acuity is at once increased to normal when air is insufflated.

An important diagnostic aid consists in the rose lustre of the promontory, which was first observed by Schwarz and which is found in many cases where the posterosuperior quadrant fuses with the postero-inferior, corresponding to the convexity of the promontory. Its origin is possibly due to pathological vascularization and hyperæmia of the mucous membrane or perhaps of the bone of the middle wall of the tympanic cavity (see p. 236). It should, however, be mentioned that a rose lustre of the promontory may also occur in perfectly normal ears.

Treatment.—Rational treatment in otosclerosis must start with the attempt at establishing the etiological factor. If a careful examination has led to the positive result of a general affection, this should receive the greatest attention. If no general affection can be demonstrated, the mode and regimen of life should nevertheless be regulated, a proper climate selected, and a suitable diet prescribed.

According to Politzer, high altitudes, sea air and bathing are not propitious, the best place to live in being one situated at a moderate altitude and in protected surroundings.

Medication.—Small doses of potassium iodide and sodium iodide (0.25 Gm. per day for 3–4 weeks, twice a year), iodipin, or Siebenmann's phosphorus medication (phosphorus 0.01 in Ol. jecor. aselli 100.0, 1–2 teaspoonfuls daily, continued for some time).

Heimann looks upon phosphorus as invigorating, improving the general nutrition, but not as improving the pathological changes of the bone. Sugár recommends phytin in the place of phosphorus. The dose for children of from two to six years is 0.25–0.50 Gm., and for children from six to ten years 0.50 to 1 Gm. daily.

According to Hammerschlag's experience in Politzer's clinic, thyroidin treatment has in no way come up to expectations. However, owing to its iodine content, it may be tried. The subjective noises are sometimes relieved by bornyval.

Any pathological changes of the nasal and nasopharyngeal mucosa should be corrected by the appropriate treatment.

Local treatment of otosclerosis through the ear-speculum is, according to Politzer, not only useless, but in some cases injurious. On the other hand, in the first stages where the motility of the stapes plate in the oval window is not yet greatly impeded, pneumomassage through the external meatus produces better results as to hearing acuity than air insufflation through the tube. This procedure, however, may not be practised for more than ½–1 minute, twice to three times weekly, and continued for about 4 weeks. After this a pause of several months must be observed.

In some cases pilocarpinum hydrochloricum seems to have a favorable effect. It should be injected through a catheter (3 or 4 drops of a

1 per cent. solution). Subcutaneous injections, which often cause manifestations of intoxication, had better be avoided in children.

Zitowitsch recommends the faradic current, introducing the button electrode deep into the tubal canal, while the second electrode is applied between the mastoid and the submaxillary angle.

Malutin reports good results from applying mud baths to the ear.

Malherbe advises "transtympanic electro-ionization," in which he distinguishes between the direct and indirect method. In the direct method one electrode (the indifferent one) is introduced into the tube, while the other is placed in the auditory canal, which has been filled with fluid. In the indirect method the indifferent electrode is applied to any part of the body; the fluid exposed to ionization is either a 2 per cent. solution of pilocarpine or, better, a 1 per cent. solution of chloride of zinc.

Ferreri's theory is to use diplococcus serum, partly on Wright's principle of vaccination and partly as instillation into the tube.

Joulin reports 10 cases of otosclerosis which were remarkably improved by X-ray treatment. Schwarz treated 3 cases in this way, in one of which—a boy 16 years old—there was an almost complete cure after six radiations, given at the rate of one per month. These favorable results may be explained by the experimentally proved arrest of new-formation of bone under the influence of the X-ray.

Although removing the stapes has been recommended, we advise against it, for the end results have not proven satisfactory owing to secondary infection or subsequent formation of adhesions.

XI. AFFECTIONS OF THE INTERNAL EAR

I. CONGENITAL ANOMALIES OF THE LABYRINTH

1. CONGENITAL DEAFNESS DUE TO THE LABYRINTH

A positive etiological factor for this very rare affection, which occurs both uni- and bilaterally, has not yet been found, although in many cases there is an hereditary taint. Either the father or mother is partially or completely deaf or there is congenital deafness in the family or in that of blood relatives. In other cases, where there is no hereditary taint as to impaired hearing, there are other hereditary degenerative stigmata. Thus, congenital deafness due to the labyrinth may be associated with trifling anomalies of the face, extremities, or trunk; or there is a general nervous taint, affections of the nerves and brain in former generations, or there may be hereditary syphilis (*q.v.*).

So far as the anatomically examined cases admit of a conclusion, the anatomical changes in this affection consist in congenital hypoplasia of the spiral ganglion and the peripheral portion of the cochlear nerve appertaining to it. The nuclei, roots, and central ramifications of the cochlear nerve, however, are perfectly intact. Corti's organ and the stria vascularis show various forms and degrees of degenerative atrophy, as in deafness of adults due to affections of the labyrinth.

Hereditary pigment anomalies also occur in congenital deafness of adults (hereditary degenerative deaf-mutism, Hammerschlag).

A classical example of congenital partial deafness is the following case which I observed and published:

A boy, twelve years old, had been slightly deaf from earliest childhood, maybe from birth. Mother and three younger children had normal hearing, but the father, then 54 years old, had been partially deaf from childhood. The father's affection had undergone no change in the course of years; vertigo or tinnitus had never occurred. Etiologically there was nothing positive to go upon; in both cases syphilis could be distinctly excluded, so that the auditory affection could only be assumed to be a congenital defect.

Examination of the boy: Both tympanic membranes showed a distinct shadow, red-yellow in color, at the border between the postero-superior and inferior quadrant, corresponding to the region of the promontory. Otherwise both tympanic membranes were unchanged. Functional test: Hearing acuity for both ears 37 feet for conversation, 27 feet for whispering, and 27 feet for the acoumeter. Weber not lateralized, Schwabach shortened, Rinné's test (c^1) positive on both sides, lower sound limit normal, upper sound limit reduced, c^4 (air-conduction) both

sides distinctly shortened. Watch positive through the cranial bones. No pathological manifestations due to the semicircular canals or vestibulum, excitability normal. no spontaneous nystagmus.

Diagnosis: Bilateral affection of the inner ear (labyrinth, auditory nerve), hyperæmia (deduced from the promontorial shadow) of the lateral wall of the labyrinth (mucous membrane and osseous capsule of the labyrinth).

Examination of the father: Both tympanic membranes normal. Functional test: Hearing acuity for conversation, right side 47 feet, left 27 feet; whispering, right side 10 feet, left 1 foot 8 inches; acoumeter, 3 feet 4 inches right and left. Weber not lateralized, Schwabach considerably shortened, Rinné's test c^1 bilaterally positive, lower sound limit normal, upper sound limit reduced, c^4 (air-conduction) considerably shortened, left more than right. Watch through cranial bone negative. No pathological manifestations of the semicircular canals or vestibulum; reflex excitability of the static labyrinth normal; no spontaneous nystagmus.

Diagnosis: Bilateral affection of the internal ear (labyrinth, auditory nerve).

According to these findings, there could be no doubt that the son's case was an hereditary-congenital affection of the labyrinth and was traceable to congenital changes (arrest of development) in the region of the nerve-ganglia, the cochlear nerve, or the membranous canal of the cochlea (Corti's organ).

The case belongs to the group of those congenital changes which, if highly developed, will lead to congenital deafness. The rarity of the case is intensified by the fact of the promontory shining through the tympanic membrane, which is ordinarily considered a characteristic sign for the clinical findings in otosclerosis. The functional test, however, proved that this was not a case of typical otosclerosis. On the other hand, recent anatomical examinations have demonstrated the fact that those changes of the osseous labyrinth capsule which show a reddish shadow also occur—though rarely—in cases of congenital deafness and cretinism; in short, in various forms of congenital ear affections. The present case, however, shows that a light congenital affection of the labyrinth may also be associated with that form of bony changes.

The rarity of these findings is explained by the fact that a slight impairment of hearing in children is easily overlooked and that these cases are not brought to the attention of the physician. The affection remains undiscovered unless it makes rapid headway at the time of puberty and leads to a higher grade of deafness. The relationship of these cases with the clinical forms of otosclerosis can no longer be doubted at the present time. Congenital anomalies of development in the region

of the cochlea and the auditory nerve no doubt represent the primordial changes in many cases of otosclerosis. To these changes are added the characteristically otosclerotic changes of the osseous labyrinth capsule, either at the embryonal or a later period.

Symptoms.—If the affection occurs unilaterally without any manifestations of the vestibular nerve, partial deafness due to the labyrinth may exist for years without any symptoms, unless it is accidentally discovered. Thus, owing to an intercurrent affection it may become necessary to examine the ear, or the child is suddenly found to be deaf if by chance the healthy ear is occluded, as may occur by lying on it in bed.

Should the affection be bilateral or associated with vestibular symptoms, it will of course be discovered much sooner. The characteristic symptom will then in most cases consist in a moderate degree of deafness. Subjective noises do not seem to occur often before puberty. Should any subjective noise be present, it is usually perceived as a very high but not intense whistling sound.

Vestibular symptoms are very rare. They consist in a slight reduction of the equilibrium, with consequent slight disturbances of gait (legs apart).

I have never observed in these cases any symptom of the semicircular canals in the shape of vertigo or vomiting. The reflex excitability of the static labyrinth was always normal.

Course and Prognosis.—The prognosis in some cases is not unfavorable, when the degree of deafness remains for life at the same low level or slightly increased. The critical age is the time of puberty. If at that period there is but slight decrease of the hearing acuity, or none at all, the prognosis remains favorable for the future. Similarly, in all cases in which the affection has occurred unilaterally and is not complicated by vestibular symptoms, the prognosis is favorable. On the other hand, in cases where the unilateral affection increases at the time of puberty, there is danger of the healthy ear becoming likewise affected.

The prognosis is unfavorable in all such cases of bilateral labyrinthine partial deafness in which the affection rapidly increases at the time of puberty and in which the history reveals a family taint in regard to congenital affections of the ear (hardness of hearing or congenital deaf-mutism). The prognosis is also unfavorable in those cases in which there are not only cochlear but also vestibular anomalies.

An improvement of the condition or a restoration of normal function is quite out of the question. In stationary cases any kind of local treatment may therefore be discarded. In progressive cases systematic galvanization of the auditory nerve is indicated, one electrode being applied close above the tragus in the mastoid fossa, the other to the nape of the neck, chest, or hand. Some authors advise application of a divided anode to both ears, with the cathode at the nape of the neck, or galvaniza-

tion transversely through the head. The galvanic current is applied 3 times a week for 5–6 weeks, after which a pause of 4–6 weeks should be observed. Besides, the use of iodine, phosphorus, iodine baths, or anti-syphilitic treatment (even in cases with negative history) is justified.

2. CONGENITAL AFFECTIONS OF THE STATIC LABYRINTH

These cases without exception furnish exceedingly interesting anomalies of reflex excitability of the semicircular canals. Thus, with an otherwise perfectly normal labyrinth the semicircular canals may be totally unsusceptible to the excitation of the rotatory chair. There is not the slightest vertigo or nystagmus after intense and long-continued rotation. But there is a positive reaction, which means positive and normal excitability, if the semicircular canals are tested by heat or electricity instead of by rotation. There are other cases in which the caloric or galvanic excitability is negative while for other tests excitability is normal.

Cases of this kind are rare and are only accidentally discovered on the occasion of other examinations of the ear. The congenital anomaly runs a perfectly symptomless course and does not cause the slightest complaint. Thus, the "galvanic insusceptibles" were accidentally discovered by Kreidl and Pollak on the occasion of the extensive investigations of these authors into the behavior of individuals with normal labyrinths on rotation or galvanic excitation.

The congenital anomalies of the static labyrinth also include over-excitability, as observed in hereditary syphilis, or abnormal excitability of the semicircular canals (*q.v.*).

In these cases there is compression- or aspiration-nystagmus, whereas a normal static labyrinth does not react upon increased or diminished air-pressure in the ear or middle ear.

The conditions for the other forms of excitability of the static labyrinth in these cases are normal, but in spite of increased mechanical excitability any one of the other forms may be temporarily or permanently diminished or destroyed. However, the positive mechanical excitability also seems to vary considerably in these cases.

Of course, the question of treatment need not be taken into consideration in these cases.

PROGRESSIVE PARTIAL DEAFNESS DUE TO THE LABYRINTH IN CHILDHOOD

Politzer was the first to report cases in which there is rapidly increasing bilateral impairment of the auditory function toward the age of 20, which sooner or later leads to considerable reduction of the same or to absolute deafness. We are indebted to Manasse for valuable contributions as to the nature of these conditions.

The functional test in these cases shows all the symptoms of an affected sound-perceiving apparatus. According to Politzer, the anatomical cause of this affection is an idiopathic atrophy of the auditory nerve. It is questionable, however, whether all cases conform to a uniform anatomical type. Part of the cases unquestionably are acquired partial deafness due to the labyrinth, but in another group of cases progressive hardness of hearing in childhood develops on the basis of a congenital labyrinthine partial deafness.

The affection is nearly always confined to the cochlea and does not spread to the static labyrinth. In an etiological respect, hereditary taint of auricular or mental affections, or other important congenital anomalies of development, as well as hereditary syphilis, should be particularly mentioned. The chief and often only symptom of the affection is a hardness of hearing, due to the labyrinth, which rapidly increases in short periods. Subjective noises occur but rarely in childhood. Examination shows a normal tympanic membrane and no changes whatever of the middle ear, admitting without any doubt a diagnosis of a middle-ear affection.

The affection occurs isolated at the auditory nerve, but may exceptionally be associated with affections of other cerebral nerves, notably the optic and olfactory.

Some of these cases belong to the domain of acromegaly, cranial deformities (oxycephalia), and the rarer affection of leontiasis ossea, in which the cerebral nerves are compressed in their canals by the osseous proliferation of the base of the skull and gradually atrophy.

Treatment.—Unfortunately, treatment is hopeless, the prognosis being in all cases unfavorable. Therapeutic success cannot be thought of. Treatment is confined to galvanization of the auditory nerve and the internal administration of iodine and phosphorus. Sometimes salvarsan injections may be indicated. In acromegaly surgical treatment of the underlying affection should be considered.

3. CONGENITAL DEAFNESS

The etiological factors are not perfectly clear. It is certain, however, that many cases of congenital (co-procreative, Hammerschlag) deafness are the expression of general physical degeneration and therefore belong to the group of hereditary-degenerative deaf-mutism in Hammerschlag's sense; nevertheless there are exceptional solitary cases of congenital deafness in healthy families with healthy parents and mentally normal, healthy children.

Hereditary taint of congenital partial or total deafness in near or remote past generations plays an important part. In these cases there is sometimes congenital deafness in several children, but it is rare that

half the number of children are so afflicted. It is rarer still that the entire progeny should be born deaf, even where the family taint is exceptionally severe. Consanguineous marriages are not conducive to pathological taints as long as both parents are physically and mentally sound and show no signs of vegetative or animal degeneration of any kind. Peiper's view is therefore to be assented to, that the progeny of consanguineous marriages between robust and healthy individuals is likewise perfectly normal and robust. Even popular tradition may be cited, according to which popular heroes—individuals who excel the normal in every respect—are supposed to have been the offspring of brother and sister or other relatives. Similarly, the breeding experiments with animals show that accouplement of healthy consanguineous animals by no means endangers the health of the young in the shape of any taint whatever.

On the other hand, the statistical investigations made by Kreidl and myself have shown that consanguineous marriages of degenerates are a very considerable factor, regardless of whether both parents or only one are affected. The degenerative manifestations of the progeny of such parents include general deformities of the body as well as of the organs of special sense, among which the hearing faculty frequently takes rank.

The taint of ear affections in the ancestors is not necessarily congenital deafness or labyrinth affection at all. Any form of congenital ear affection, with otosclerosis in the forefront, and exceptionally even anomalies of development of the auditory canal and concha (atresia or other defects) in the ancestors, may be followed by congenital deafness or other congenital defects of the labyrinth in the immediate or remote offspring in future generations.

Anatomy.—The anatomical foundation of any form of congenital deafness lies in pronounced anomalies of the cochlear nerve and its peripheral end apparatus, Corti's organ.

In most of these cases there is an originally defective embryonal rudiment of the nerve-ganglia of the cochlea and Corti's organ (congenital hypoplasia of the cochlearis). In other cases the embryonal rudiment of these parts is normal, and there will be only secondary atrophy of the nerve-ganglia and a degeneration of Corti's organ, owing to defective or arrested development of the auxiliary apparatus of the cochlea and the rest of the ear (capsule of the labyrinth and tympanic cavity).

Accordingly, two groups of congenital deafness may be distinguished:

I. Congenital deafness from congenital aplasia or hypoplasia of the cochlear nerve, the spiral ganglion, and Corti's organ.

II. Congenital deafness from atrophy of the cochlear nerve, spiral ganglion, and Corti's organ, owing to congenital defective or arrested

development in the area of the capsule of the labyrinth or of the middle ear. The comprehensive labors of Siebenmann and Denker are of great importance in relation to the classification of the various findings.

I. In Group I there are the following subdivisions:

(1) The congenital anomaly of the embryonal rudiment of the labyrinth may be confined to the cochlea. From this results the type of sacculo-cochlear degeneration:

Semicircular canals and utriculus normal; the congenital anomaly of the labyrinth is restricted to the pars inferior. This type is found in about 70 per cent. of the congenitally deaf. The functional test shows normal semicircular canals and no equilibrial disturbances.

The functional test methods of the vestibulum are not yet sufficiently advanced to allow of the recognition of the functional insufficiency of the macula sacculi as compared to the function of the intact macula utriculi. In about one-third of these cases circumscribed areas of Corti's organ and its apparatus of nerve-ganglia are functionally efficient, so that remnants of hearing ability may be observed.

(2) Congenital hypoplasia comprises the entire apparatus of nerve-ganglia and the nerve-end places of the whole labyrinth. In these cases the entire eighth nerve in the internal auditory duct is as thin as a thread; all the six nerve-end places of the labyrinth are merely indicated by accumulations of prop-cells or are entirely absent. In these cases there is complete deafness and absent excitability of the static labyrinth, combined with congenital equilibrial disturbances of the labyrinth.

(3) Aplasia of the entire membranous labyrinth. This is of rare occurrence and of subordinate clinical importance. It is always a part manifestation of pronounced malformation of the brain and head (anencephalia, cyclopia); it may also be found in double monstrosities, in short in individuals who are either non-viable owing to other congenital defects, are stillborn, or will die within a few hours after birth.

In all three forms of congenital deafness there are more or less important changes in the shape of the membranous labyrinth, aside from the anomalies described.

Obliteration of the pars inferior of the membranous labyrinth is a typical condition in the sacculo-cochlear type. The hollow spaces of the membranous labyrinth may be either constricted or dilated. Furthermore, abnormal embryonal folds have formed membranous septa which traverse the hollow spaces of the labyrinth or through which free parts of the membranous labyrinth may be encysted (membrana tectoria, otoliths).

The defective development of the sensory epithelium is, of course, associated with considerable epithelial metaplasias. In nearly every case belonging to this group there are large quantities of abnormal, bizarre

forms which in most cases belong to the prop-cell type, less often to the hair-cell type, aside from the few remaining normal epithelial cells.

The totality of the degenerate combined epithelial cells of the membranous labyrinth may develop into pathological cell heaps which either remain attached to the membranous wall or are encysted in a double septum, or are freely suspended in the endolymphatic spaces.

A typical accompanying manifestation of aplasia or hypoplasia of Corti's organ is aplasia or atrophy of the stria vascularis.

II. Congenital deafness from atrophy of the cochlear nerve, spiral ganglion, and Corti's organ, owing to congenital defective or arrested development in the area of the capsule of the labyrinth or of the middle ear. To this group belong:

(1) The anatomically interesting deformity of the membranous cochlear canal resulting from insufficient development of the osseous cochlear capsule. This type of congenital deafness in man was first demonstrated by myself, and in animals by Tandler and myself,—not by Mondini, as Siebenmann erroneously states. The embryonal membranous cochlear canal is first covered laterally by a cartilaginous crust, which gradually deepens and finally assumes the external shape of the later cochlea. A crest, which separates the vestibular section of the cochlear from the cochlear body, rises from the interior surface of this cartilaginous crust.

Should the development of the cochlear capsule be arrested at this stage and lead to ossification, the other scala will fail to develop. The scala vestibuli will then communicate with the scala tympani of the next higher convolution, and the membranous cochlear canal will assume an entirely abnormal shape and position. In that case there will be abundant cell material in the area of the nerve-end places and their auxiliary apparatus, and the impression given by the histological picture is that the final anomalies of Corti's organ, the stria vascularis, and the membranous cochlear canal have developed at a later period, and as subsequent manifestations of the arrested development of the capsule of the labyrinth.

(2) Pathological osseous foci in the capsule of the labyrinth, combined with degenerative atrophy of the nerve-end places of the labyrinth.

The cases belonging to this group have been elucidated by the findings of Lindt, Manasse, Politzer, Siebenmann, and myself.

The membranous labyrinth has a more or less normal shape, or there is congenital hypoplasia or aplasia of one or both vestibular sacs, the canalis reuniens, and the vestibular cul-de-sac. All the nerve-end places are atrophied, and the hair-cells are absent. There are pathological osseous foci in the capsule of the cochlea of the type of osteitis vasculosa. They entirely conform to those foci which characterize the

anatomical findings in otosclerosis. In some cases there is also found constriction or osseous occlusion of the labyrinth windows and obliteration of the cochlear aqueduct.

The pathological osseous foci are situated in the lateral wall of the labyrinth, in the neighborhood of the windows of the labyrinth, in the promontory, or deep in the petrous bone. Siebenmann found osteitic osseous foci in the area of the semicircular canals and vestibulum, Manasse in the region of the internal auditory duct, and myself in the region of the internal auditory duct, of the basal wall of the cochlea, and of the vestibulum in a congenitally deaf cretin.

This type of congenital deafness is a stepping-stone to the various forms of congenital hardness of hearing. There can be no doubt, in the light of our present experience, that in otosclerosis, too, the first rudiments of the pathological osseous foci are often of congenital origin, and that other changes of the capsule of the labyrinth, the ankylosis of the stapes, and the final destructive atrophy of the sensory epithelium are secondary, postembryonal defects. No doubt, congenital osseous foci of this description may exist more or less without symptoms until puberty, and only then give rise to serious functional disturbance.

(3) Congenital anomalies of the middle ear, combined with atrophy of the sensory epithelium of the labyrinth. This type of congenital deafness is best characterized by Siebenmann's findings. There are considerable anomalies in the chain of auricular ossicles, combined with atrophy and epithelial metaplasias of the membranous labyrinth.

According to the findings in adult deaf-mutes, the perilymphatic tissue is usually atrophied and surprisingly poor in substance.

(4) Congenital atresia of the middle ear is only rarely combined with congenital deafness. The most marked arrest of development in the majority of these cases occurs in the external and middle ear, without interfering with the development of the labyrinth. The functional test in patients with congenital atresia of the external auditory meatus shows in most cases normal function of the labyrinth.

A. CLINICAL OBSERVATIONS ON CONGENITAL AND ACQUIRED DEAFNESS AND DEAF-MUTISM

Examination of these cases reveals either a normal or slightly changed tympanic membrane. Catarrhal changes of the middle ear and adenoid vegetations are not infrequent. Congenital anomalies of development of the concha or external auditory meatus are only exceptionally present in congenital deafness.

The functional test of the internal ear is most important.

It is to be found out whether deafness is complete or only partial. In the latter case the functional remnants must be determined in regard

to both quantity and quality. Itard's system of dividing the material in deaf-mute institutes into five groups is still the best for the quantitative determination. These groups are:

(1) Deaf-mutes with a hearing acuity of 6 feet 8 inches for conversation.

(2) Deaf-mutes with retained perception of loud speech close to the ear.

(3) Deaf-mutes with preserved vowel perception.

(4) Deaf-mutes with preserved perception of loud noises (rattle, trumpet, whistle, hand-clapping, key-rattling, clock, alarm clock).

(5) Total deaf-mutism (no functional remnants).

The question whether there are any functional remnants at all can be determined in infants as young as six months, provided they are otherwise normal. The quantitative determination, according to the above five groups, is only possible at a higher age. Thus, for group 3 the lowest limit is three years; for groups 1 and 2 the limit is 4–5 years. As to groups 1 and 2, children should have reached school age and perhaps have had some education either at home or at an institution for the deaf-mute.

Should deaf-mutism be complicated by idiocy, it is difficult and sometimes impossible, at any time of life, to diagnose whether there are any remnants of hearing ability.

It is advisable to test each ear separately, closing the other ear with the finger or a cotton plug saturated with glycerin. In timid children the unilateral occlusion may be dispensed with; instead, their attention should be diverted from the test by some toy given them to play with. The first part of the test would be to observe whether a sound or noise causes a motor reaction. The slightest degree of reaction consists in the child's eyes turning in the direction of the sound. A more decisive reaction consists in the head or body being turned, and the strongest reaction, which consists in starting up with a muscular contraction, can only be obtained by very powerful noises, unless there are considerable hearing remnants.

In order to make a safe diagnosis, it is necessary that the child should react regularly upon the respective sounds, and that modifications of the test should exclude errors due to tactile or optic sensations or suggestions. The examiner should stand behind the child without touching him, also taking care that the air current from the instruments used (rattle, whistle, trumpet) is not directed toward the body of the patient, lest the sensation cause a motor reaction without any perception of sound.

If hearing remnants are to be determined in deaf-mutes who have already had lessons in lip-reading, the possibility of indirect reading should be reckoned with. The following is an interesting case in point:

An intelligent boy eight years old was transferred to a new deaf-mute

institution, where a new examination for hearing was made. His face was turned toward the door, and the examiner, standing behind his back, was surprised by the considerable response he displayed, which could not have been expected considering his defective articulation. The explanation was forthcoming in a few minutes. The child observed the movements of the examiner's lips from the reflection in the highly-polished door and was thus able to read off the words indirectly. He did not have the slightest hearing remnants.

A qualitative determination of any hearing remnants can, as a rule, only be made after the child has had some lessons in an institution. The examination consists in the monaural test with the hand-organ (harmonica) devised by Urbantschitsch or with Bezold's continuous tuning-forks, the object being to determine whether there are hearing remnants between b^8 and g^2. Should this be the case, even though partly or slightly, the child can be instructed by the ear in the so-called hearing classes, with a fair chance of success. In some cases this test cannot be reliably made until the end of the first school year, in others not before the second or third year.

The examination should be distributed over several sittings, as the child becomes easily fatigued and the entire examination occupies from one to two hours.

The qualitative determination of any hearing remnants is only of practical value in institutions which have hearing classes, because here the hearing ability of the child is utilized, while in the other classes nothing but articulation and lip-reading is practised. As a matter of course, the lessons can be assimilated much quicker in hearing classes and a higher degree of learning be imparted.

There are model hearing classes after Bezold at the deaf-mute institutions of Munich and Weissenfels.

The presence of hearing remnants may also be deduced from the gait of deaf-mutes. Those totally deaf or with a nonfunctioning vestibularis drag their feet, when wearing shoes, causing a grating noise. They cannot hear the noise themselves, and, as the stability of their body is impaired, they are compelled to feel their way by a dragging manner of walking. Should, however, any hearing remnants be present, patients will perceive the noise they cause and avoid it.

A second important sign is articulation. The sounds emitted by those totally deaf are always clumsy and inarticulate; they can only be produced with the aid of tactile sensation and optically acquired positions of the mouth and tongue. They know the intensity of the sound by the volume of air consumed, which they have learned to estimate by practice. They will be in a fight with their tongue to a certain extent for the rest of their lives, and will rapidly fatigue from using it.

Should, on the other hand, the slightest serviceable quantitative or qualitative remnants exist, deaf-mutes will make themselves understood, articulate cleverly and find something like correct intensity and modulation in conversational language, especially if any hearing remnants for words have been preserved.

One should, therefore, never neglect to pay attention to the ordinary gait and speech of the deaf-mute when determining the presence of hearing remnants.

The clinical examination of the static labyrinth of deaf-mutes has a certain value for the differential diagnosis.

Most of those congenitally deaf-mute possess semicircular canals of normal reaction, and also a normal utriculus. The congenital affection is restricted to the pars inferior of the labyrinth, to the cochlea and sacculus.

Two considerations will serve to explain this clinically established fact:

Two organs of very different phylogenetic age are united in the membranous labyrinth. The static labyrinth—*i.e.*, the vestibulum and the semicircular canals—is present down to a very low class of animals. Even the invertebrate animals possess a static labyrinth (statocyst) and in some plants cellular organs can be recognized which are homologous to the animal static labyrinth.

The cochlea, on the other hand, is found only in animals of a higher degree of development, going upward from the amphibia, reptiles, and birds to the mammals.

Now it is a well-known experience that the resisting power of an organ increases with its phylogenetic age, and it can, therefore, be well understood that a congenital injury leaves the static labyrinth unchanged, while it arrests the development of the cochlea.

The second consideration arises from a histological examination of the nerve-ganglia. The nerve-ganglia of the cochlear nerve consist of considerably smaller cells and thinner nerve-fibres than those of the vestibularis, and the power of resistance to injury is the slighter, the tenderer the elements of which an organ is composed.

Acquired Deafness.—Most cases of acquired deafness may be anatomically traced to inflammation of the labyrinth. The pathological factor exerts such an intense effect that usually the entire labyrinth (static and acoustic) perishes from the inflammation.

The typical functional findings of acquired deafness, therefore, are: Total deafness, without any hearing remnants, and absence of excitability of the static labyrinth. In congenital deafness, on the other hand, the typical functional findings are preserved function of the static labyrinth with preserved hearing remnants in a considerable percentage of cases.

Should the history be doubtful, congenital deafness can with certainty be assumed from preserved excitability of the static labyrinth, and with great probability from preserved hearing remnants.

B. DEAF-MUTE STATISTICS. EDUCATION OF THE DEAF-MUTE

1. The Number of Deaf-mutes in the Various Countries

Excellent and exact statistical investigations have been made throughout Germany in conjunction with the census of December 1, 1900. The results were classified and published by the Imperial Board of Health. According to these figures, the total number of deaf-mutes was 48,750, of whom 54.1 per cent. were male and 45.9 per cent. female. The prevalence of deaf-mutes in thinly populated country districts can be explained by the fact that the conditions of life and the hygienic facilities are less favorable there than in the cities or in districts which, owing to great fertility or favorable economic conditions, can offer better nutrition and hygienic protection. Of the total number mentioned, 75 per cent. were congenital and 25 per cent. acquired.

As to Bavaria, there were 5281 deaf-mutes, 52.6 per cent. of whom were male and 47.4 per cent. female.

A census in Austria at the end of 1905 showed 15,303 male and 12,264 female deaf-mutes,—in all, 27,567. This shows a reduction by 300 as compared with 1904. Practically the same provinces in which cretinism is most prevalent have the greatest number of deaf-mutes: Carinthia, Salzburg, and Styria.

Galicia has many deaf-mutes and few cretins, whereas in upper Austria and the Tyrol the reverse is the case.

In Italy there are 38.8 deaf-mutes to every 10,000 inhabitants.

In France there were 19,579 deaf-mutes in 1900.

Belgium, with a population of 6,693,180, had 3500 deaf-mutes.

In Spain there is 1 deaf-mute to every 2000 inhabitants. The total figure given of 7639 seems too low; 60 per cent. of these are said to be congenitally deaf.

In Russia the total number of deaf-mutes has been estimated at more than 200,000.

In 1894 a census of deaf-mute children was taken in Livonia. Result: 61 below 8 years, 187 between 8 and 13, 819 above 13.

Sweden registers 110.9 deaf-mutes to 100,000 inhabitants. Total number in 1890, 5307.

In Norway there were 1176 deaf-mute men and 963 deaf-mute women in 1891; 1690 were congenital, 449 acquired; 514 were below the age of 15.

Denmark, with a population of 2,200,000, has 1400 deaf-mutes, or 60 per 100,000.

In Finland there were 2767 deaf-mutes in 1890—1537 male and 1230 female.

Switzerland has 899 deaf-mute school-children.

Holland has relatively the smallest number of deaf-mutes.

The number of deaf-mutes in the United States according to the census of 1900 numbered 89,287.

2. Number of Deaf-mute Institutions

Austria	26	Hungary	8
Belgium	12	Italy	47
Canada	7	Luxembourg	1
Denmark	3	Norway	5
England	70	Russia	27
Finland	8	Spain	11
France	67	Sweden	7
Germany	91	Switzerland	16
Holland	4	United States	148

Most of these institutions are equipped for resident patients, but they also treat outside patients. They usually admit boys as well as girls, but the education is mostly separate. Unfortunately, a large number of these institutions are still conducted in conjunction with blind and orphan asylums.

3. Number of Pupils Instructed Annually

Austria	1,938	Hungary	550
Belgium	1,265	Italy	2,519
Canada	768	Luxembourg	22
Denmark	348	Norway	309
England	4,222	Russia	1,350
Finland	365	Spain	475
France	4,098	Sweden	803
Germany	6,497	Switzerland	723
Holland	504	United States	12,721

In the German statistics for 1902 and 1905, 6689 question blanks regarding the degree of hearing remnants of 53 German institutions have been incorporated (3130 congenital, 3123 acquired, and 436 undecided cases). The following is a summary of the collected material:

					Hearing remnants.			
	1	2	3	4	5	6	7	8
Born deaf	43.1	11.8	1.0	8.0	13.0	2.9	8.2	12.0
Become deaf	46.7	13.5	1.7	8.2	12.0	2.0	6.2	9.5
All, including 436 doubtful	44.4	12.8	1.4	7.9	12.7	2.5	7.5	10.9

1. Hearing entirely destroyed on both sides.
2. Sound tested, but result not stated.
3. Undecided sound-perception.
4. Sound-perception.
5. Vowels.
6. Consonants, except p, t, r, k.
7. Single words.
8. Sentences.

Among the deaf-mutes in the German institutions 50.4 per cent. showed deafness in "earliest childhood" and 49.6 per cent. "later." This nomenclature is by no means equivalent to congenital and acquired.

4. Kindergartens and Preparatory Schools for the Deaf-mute

In Berlin there is a Society for the Erection and Maintenance of Kindergartens for Deaf-mute Children between the Ages of 3 and 7 Years. The first kindergarten was opened April 1, 1894, with 8 children (later 14). The program was principally to impel children's attention by appropriate plays and occupation. No attempts are made at imparting speech. Much weight is attached to the nature of the occupation, physical care and education. The institution is under medical supervision and direction. Individual medical examination is directed to constant observation of the hearing ability, speech and physical conduct.

In England it has been possible by private donations to equip all deaf-mute institutes with classes for children under school age. Children under 7 years are also instructed in special preparatory classes of the day-schools.

In Sweden deaf-mute children are admitted to the kindergarten of hearing children after the system of Bell, of Washington, but, besides, receive a few hours' separate instruction daily by a special female teacher. A preparatory school for deaf-mutes exists in Gothenburg, where a trained deaf-mute teacher prepares children between the ages of 3 and 8 years for the district school at Wenersborg.

In Moscow a kindergarten for deaf-mutes has existed since 1900, in which children from 4 to 7 years old receive training and education.

In France there are preparatory classes (classes enfantines) in the government deaf-mute institutes of Paris, Bordeaux, and Dijon.

In Vienna the opening of a kindergarten for deaf-mutes who have attained school age is contemplated by the Imperial Deaf-mute Institute.

5. Compulsory School Attendance

In Germany and Austria deaf-mute children are included in the general law for compulsory school attendance. The number of deaf-mute institutes in Germany has increased to such an extent in the course of years that some of the federal states were in a position to accord instruction and education to all deaf-mute children who are capable of following the lessons. It so happened, however, that the parents either opposed these humanitarian provisions or were indifferent to them, and consequently some of the states have found it necessary to introduce compulsory attendance. The deaf-mute teachers have demanded compulsory attendance for years, at the same time furnishing proof by publications in their special journals of the disadvantages which would accrue from non-compulsory attendance.

In England every deaf-mute child is compelled to attend the day-school up to the sixteenth year. All city schools for deaf-mutes—that is, day-schools which are conducted by public committees—furnish instruction and education free of cost. For resident children or those assigned to foster-parents, the relatives contribute a sum according to their means. If they show that they are without means, both education and expense of living are furnished free. England, therefore, is the country which takes the best care of its deaf-mute children. They are not admitted to ordinary public schools.

In Sweden a special law for the instruction of deaf-mutes has existed for a long time. This law applies to (1) children who were born deaf or became so at an early age; (2) those who have become deaf after having learned to speak, but require instruction by deaf-mute teachers to prevent their becoming mute; (3) mentally defective deaf-mutes who cannot be instructed in a school for deaf-mutes. The object of the entire law is compulsory school attendance of all deaf-mutes. Children are compelled to attend school for 8 years, but on furnishing proof of adequate knowledge may be discharged earlier.

As to Switzerland there are no generally applicable legal provisions for compulsory school attendance. The Canton of Berne, however, has a school-law to the effect that deaf-mute children capable of being taught, but unable to receive instruction in the public schools, will be assigned to special institutions or classes which are maintained by the state.

Extensive provision for the instruction and training of all deaf-mutes has been made in the United States and Canada.

Institutions for resident inmates preponderate in all the states, and it is only in this way that an undisturbed progressive instruction can be imparted. It is an important modern demand that the instruction be not arranged on too large a scale. An institution should not have more than 150 inmates. Boys and girls can be co-educated, but the various classes should not have more than 8–15 pupils each.

There are a much smaller number of non-resident institutions, and the arrangement is here for children to be at home outside of school time. The resident institutions likewise recognize the high value of family education, and accordingly allow vacations at Christmas and Easter, during which the children are discharged for home. In selecting a deaf-mute institution it is also an important point to see that the child receives instructions in his native language.

6. *Advanced Education for Deaf-mutes*

An excellent school for advanced education for deaf-mutes is attached to the Imperial Deaf-mute Institute at Vienna. Instruction is here given in the evening and on Sunday mornings. Its object is to fortify

deaf-mutes in the knowledge they have already acquired and to equip them with such further knowledge as they will require for the practical purposes of life. The most important subjects taught are: drawing, commercial arithmetic, single-entry book-keeping, writing letters and business documents, economics. The advanced school is principally attended by those who, during school age, have already had instruction at the Imperial Deaf-mute Institute. In this way the sense is fostered of belonging together and of forming a bond of union which is a necessary adjunct to the success of the deaf-mute.

There are similar schools for advanced education established by the Vienna Aid Society for Deaf-mutes and the Deaf-mute Institute at Prague.

In Germany schools for advanced education are attached to a great number of deaf-mute institutes.

In France schools for deaf-mute apprentices (écoles professionelles) are attached to the deaf-mute institutes of Albi, Arras, Besançon, St. Cloud, Dijon, Elbeuf, Caen, Chambery, Limoges, LePuy, St. Etienne, Toulon, and at the National Deaf-mute Institute of Paris. In these workshops boys receive a thorough training. In Paris, for instance, the boys are instructed in horticulture, shoemaking, tailoring, printing, and wood-carving. An asylum with workshops for girls, who cannot return home to their families after leaving the school, exists in Bourg-la-Reine.

In Belgium all inmates of deaf-mute institutes are taught skilled labor. The instruction commences at the age of 13. Previous to learning the actual work, pupils are trained in Froebel work and modelling. In the selection of the work the wishes of parents and guardians are considered, but chiefly the aptitude and health of the boys and the conditions under which they will have to live later on.

Spain seems to attach much weight to the professional education of deaf-mutes. In Madrid they are instructed in printing, bookbinding, shoemaking, wheelwright and locksmith work. The institutions at Bourgos and Seville are conducted on a similar plan. The tailors and shoemakers seem to make a fair living there, as special workshops for this kind of work are attached to the institutions. In the non-resident institutions of Barcelona, Valencia, and Saragossa, technical drawing and needlework for women are favorite subjects.

England has evening schools aside from the day schools. They are attached to the various institutions as advanced classes and are maintained by donations and government contributions. The subjects taught are the English language, wood-carving, cooking, and ladies' tailoring. There are also missions distributed all over England to aid and support adult deaf-mutes. Their special object is to promote the religious, social, and domestic life of the deaf-mute. Some of the deaf-mute societies

have made it imperative for their members to continue the sound language learned in the schools.

Russia has appointed special workmen's teachers at the deaf-mute institutes. Pupils receive their technical training during school age. Warsaw has a Sunday-school for deaf-mute workmen. Moscow has an asylum for female deaf-mutes above school age and an aid society for adult deaf-mutes.

In Denmark deaf-mutes are closely controlled both before and after school life in regard to occupation, marriage, and children. There are no special schools for advanced education.

In Finland a number of advanced schools are maintained by the teachers for the benefit of their former pupils. Instruction is given during the vacations. A few deaf-mute societies have also arranged for advanced education for deaf-mutes.

The institute at Groningen, Holland, is in the habit of sending to its former pupils so-called open letters with a view of keeping in touch with them after they have entered practical life. There are no advanced schools, but some such arrangement has recently been planned.

An advanced mental education is accorded to deaf-mutes nowhere except in America, where pupils are even enabled to have college education. Advanced schools exist in nearly every large city in America and are closely connected with the excellent organizations there for adult deaf-mutes.

7. *The Choice of an Occupation for Deaf-mutes*

It is important to see that deaf-mutes learn a simple business which enables them to make a living and furnishes them with full occupation all the year around. Deaf-mutes, therefore, work as shoemakers, tailors, saddlers, bookbinders, gardeners, compositors, metal-workers, basket-makers, inside painters, lithographers, decorators, engravers, ornamental carpenters, sculptors, wood-carvers, etc. Conditions are less favorable for girls than for boys. They are generally occupied as seamstresses, tailoresses, milliners, ironers, flower-makers, cigarette-makers, or domestics. Both boys and girls do well in agriculture.

When the instruction is completed, most of the deaf-mutes return to their native places. At a few Austrian institutes there is a special fund out of which poor apprentices are furnished with tools and clothing. At the places where there are institutes there are usually a number of resident employers who willingly take in apprentices in appreciation of their good ethical and physical qualities (attachment, industry, and manual dexterity).

Pongratz has compiled statistics on the occupations and earnings of deaf-mutes in Bavaria, and thinks that those with a good training

will migrate into the cities where a greater number of them can find independent positions, as in regard to capability and earning power they are but little behind those in full possession of all their senses.

In Belgium there are special instructors for nearly all occupations. If any particular occupation is not provided for, the directors of the institute send applicants to proper places where it can be learned.

In England deaf-mutes, aside from many other occupations, are often employed by the post-office as sorters. They are excluded only from such work as might endanger them on the ground of deafness.

In all Italian deaf-mute institutes there are classes for intellectual as well as industrial instruction. The former is based on the principle of verbal instruction; the latter depends on the future occupation.

In Russia the pupils are left to themselves after school age. At the request of parents, however, the teachers find places for their pupils with some employer. A large number take up agriculture. At Mitau every deaf-mute was formerly compelled to become a tailor or a shoemaker, but fortunately this compulsion is now abandoned.

In Finland most of the male deaf-mutes work on farms or in factories; the female ones work in tobacco or candy factories.

In Switzerland the deaf-mute authorities object to the pupils going to the large cities and endeavor to find them employment in the neighborhood of their native places.

In Holland there is compulsory education for both intellectual and industrial occupation, which is usually given in large workshops attached to the institutes. The instruction includes such occupations as tailors, shoemakers, carpenters, turners, vat-makers, printers, farmers for garden and field, but not all this at every institution. Girls learn ordinary and fancy needlework, measuring, cutting, and operating the sewing-machine. Those with particular aptitude are instructed in applied art, older ones in ironing and cooking. The Groningen report for 1900 has the following in regard to the earning power: "Although it is not to be expected that all discharged pupils will do equally well in the struggle for existence, yet most of them have been equipped sufficiently to earn at once part of their living."

8. Provision for Deaf-mutes by Public Charity or Government Help

At the end of 1900 there were in Germany 12 homes for deaf-mutes, the first object of which is to provide for poor old deaf-mutes who are unable to work. The care for the education of the deaf-mute devolves upon the provincial administrations. It was a gratifying advance in the development of the care for the deaf-mute in Germany when the provincial administrations took over the institutions. The number of institutions was greatly increased, a large number of new buildings were

erected, the time for education was prolonged, the industrial training of the pupils was improved, and the salaries of the teachers were increased.

In Bavaria, according to the census for 1900, 491 deaf-mutes had been placed in homes (9.3 per cent. of all the deaf-mutes in Bavaria).

In Austria the deaf-mute institutes are subordinate to the Imperial Ministry for Education in a pedagogic respect, but in all other respects they are subordinate to the various local authorities who see to their maintenance. There are no homes in Austria as yet for deaf-mutes, but there are deaf-mute societies in Vienna, Graz, Lemberg, Prague, Salzburg, which foster humanitarian objects.

Of the 27,576 deaf-mutes living in Austria in 1905, 131, or 0.5 per cent., had been provided for by charity.

In Italy the deaf-mute institutes of Genoa, Milan, Naples, Palermo, Rome, and Siena are maintained by the state.

In Belgium the institutes for the deaf-mute and the blind are subordinate to the Ministry of Justice as benevolent institutions. There are 4 asylums for female deaf-mutes (Brussels, Bruges, Ghent, Namur). The one at Namur also admits uneducated female deaf-mutes, also mentally afflicted and poor female orphans, the sick, and those who wish to enter a holy order. At Antwerp, Brussels, Charleroi, Ghent, Liège, Namur, and other cities there are societies for mutual aid, finding work, refuges, etc. All these societies formed a federation in 1901 at the III National Congress at Lourrain, through which the well-being of the deaf-mute is to be promoted.

The local authorities in Belgium are obliged to send poor deaf-mute children into a special institute and to leave them there until they have attained mental and industrial efficiency. At the age of 65 every poor deaf-mute is entitled to a pension of 65 francs a year.

The English institutions are maintained out of state and private means.

The Russian institutions are chiefly dependent on private donations and exist usually as society institutions. Homes for the deaf-mute do not exist as yet in Russia.

In Sweden a number of aid societies have been formed by adult deaf-mutes, among which the one at Stockholm is the largest and richest. The institutions themselves in Sweden and Norway are maintained out of state and private means.

In Denmark there are several homes for adult deaf-mutes who are unable to work and for old men.

In Finland a society for the care of the deaf-mute comprises the whole country.

In Switzerland there are no homes whatever for the deaf-mute, but the institution at Zurich has been left a legacy to establish one,

so that probably Zurich will have the first home for old deaf-mutes unable to work.

The Swiss institutions are maintained out of public and private means.

In Holland public charity almost exclusively supplies the needs of the deaf-mute.

A large number of humanitarian institutions provide for the needs of adult and old deaf-mutes in America. There are also special libraries and evening schools for adult deaf-mutes.

9. Education and Provision for the Deaf-mute Blind

As to Germany, Riemann, teacher at the Royal Deaf-mute Institute, deserves credit for what he has done in the interests of the blind deaf-mute. There are homes for the blind deaf-mute at Nowawes and Ketschendorf, near Fürstenwalde.

In Sweden there is a school home for blind deaf-mutes, which was erected in Skara in 1886 and transferred to Wenersdorf in 1892. Among the pupils a boy and a girl have shown unusual intellectual development which bears comparison to that of the well-known Laura Bridgman and Helen Keller. The instruction was given by the ordinary hand alphabet method. Having thus been introduced into an understanding of the language, they also learned to speak. Aside from the blind deaf-mutes, there are also several mentally afflicted and blind children at this institution, which receives a large yearly contribution from the state.

The department school at Hamar, Norway, reports a few successful results in the education of blind deaf-mutes.

The case of Helen Keller, of Boston, is widely known, as, thanks to an exceptionally gifted teacher, she has attained a high degree of culture.

C. DEAF-MUTE INSTRUCTION

Both congenital and acquired deaf-mutism are in a general way preëminently a disease of the poor. The active factors which lead to congenital deafness are in many cases furnished or favored by the poverty of the parents. In acquired deaf-mutism the poverty of the family is a still greater factor. It is well known that cerebrospinal meningitis occurs oftener among the poor than the rich classes. In acquired deafness resulting from scarlet fever, measles, etc., poverty again plays a rôle, since with good hygienic precautions and timely competent treatment most acute infections run their course without any ear complications, or they are confined to the middle ear, and a spreading of the inflammation to the labyrinth is hardly to be apprehended. The hygienic precautions also include the timely removal of the faucial tonsils. Of course, we know that children with considerably increased lymphadenoid

tissue in the nasopharyngeal space are more prone to contract ear inflammations in the course of acute infections than children with a free passage in the nasopharynx.

The deaf-mute child can have his school education privately in his family or as a resident or non-resident pupil of a deaf-mute institute. Owing to extraneous circumstances, the majority of deaf-mute cases must be admitted to institutions as resident patients.

The deaf-mute child is placed in the institution at the age of seven or eight years, where he will remain, with the exception of vacations, until he has completed his eighth school year. It seems that the institutions answer their purpose best if they do not admit more than 150 pupils.

The entire program of instruction also makes it desirable that a class should not consist of more than from eight to fifteen pupils. Instruction in a deaf-mute institute is more effective the closer it approaches personal instruction. All the pedagogic demands in regard to the consideration of the individuality of the pupil, which are of the greatest importance even for normal pupils, gain in importance when children with only four senses are concerned. The instruction consists in teaching lip-reading and articulation, and with these methods the subjects taught in public schools are mastered in the course of eight years.

At the same time, sign language as a substitute for speaking is learned and used, but there is no regular instruction in this subject. New pupils readily learn the same from the older ones, as the child prefers using it at play and at work rather than making sounds. Recently it was suggested to give systematic instruction in gesticulation. The great practical use deaf-mutes can make of it, especially in conversing with each other, seems to lend weight to the suggestion; but it cannot be denied that, as compared with instruction in lip-reading and articulation, sign language is of secondary importance. The understanding of higher conceptions, the formulation of conceptions, and the ethical development of the deaf-mute children, alike are inseparably bound up with the method of articulation and lip-reading. By using pure sign language it is impossible to impart higher conceptions to the child. Nevertheless, the suggestion of Kümmel and Passow to give systematic instruction in preparatory schools to be newly founded is a salutary one, as instruction in articulation can be built upon the knowledge acquired by the sign language.

Urbantschitsch has repeatedly emphasized the importance of hearing exercises. These exercises, for which he has arranged methodic instruction, are intended to initiate the understanding of the auditory impressions received, to help explain their meaning, to stimulate attention for the various auditory perceptions, and to increase the sound-perception.

Urbantschitsch also recommends to extend instruction by the ear only to those deaf-mute children who possess relatively large hearing remnants, but disagrees with Bezold that those unable to hear the tuning-fork from a^1 to b^2 should be excluded from the hearing lessons.

Bezold's suggestion to inaugurate hearing classes has borne good fruit. Only such pupils as are provided with hearing remnants qualitatively and quantitatively sufficient to follow the instructions by the ear are admitted. It is advisable to place pupils from various institutions whose hearing properties are the same together in one class, as it is unlikely that one single institution has enough material to form such classes. The success which has attended the hearing classes is very satisfactory, as the subjects can be mastered quicker in these classes and higher instruction imparted. Of course, it has always been known that articulation in children with serviceable hearing remnants is incomparably better than that of deaf-mutes.

Hartmann's auxiliary schools occupy a medium stage between the hearing classes and normal schools. These schools are intended for children with partial deafness whose hearing ability is not sufficient to follow instructions in normal schools (congenital deafness under 6 feet 8 inches, acquired deafness under 1 foot 8 inches, loud language, provided the child has been able to speak before and has not yet completely unlearned it). According to Hartmann's statistics, it would be necessary to open schools for children with partial deafness in all cities of 150,000–200,000 inhabitants.

The care for the deaf-mute before school age still leaves much to be desired in Europe. In America the deaf-mute institutes before the school age do excellent work. They are conducted on the principle of the German kindergartens. Parents are entirely relieved of the care for the deaf-mute children. The opening of similar institutes in Germany at an early date is urgently to be wished for. Kindergartens for deaf-mutes would facilitate later instruction in the institutes not only in a somatic, but also in an intellectual respect. In the kindergarten itself, at play and at work, deaf-mute children preferably use the sign language to converse with each other, but under the guidance of competent teachers they will acquire a thorough knowledge of lip-reading and articulation.

II. INFLAMMATORY AFFECTIONS OF THE LABYRINTH

Etiology.—Inflammation of the labyrinth may occur in the course of general affections, poisoning, or traumatic injury to the head or ear; as a rule, however, it is caused by some inflammation in the neighborhood (middle ear, endocranium) spreading to the labyrinth.

According to their origin, suppuration of the labyrinth is divided into meningitic and otitic forms. The former occurs by the spreading

of a suppurative process from the brain and meninges to the labyrinth; the latter, by the spreading of a middle-ear affection to the internal ear. The involvement of the labyrinth itself occurs either by direct spreading of the suppuration by continuity or by metastasis. Suppuration of the labyrinth which has developed in the wake of endocranial affections is always caused by continuity of process. Otitic suppuration of the labyrinth, however, may have been caused by direct spreading of an infection as well as by metastasis.

Anatomy.—The suppurative inflammation in the hollow spaces of the labyrinth itself is designated empyema of the labyrinth, unless the surrounding bone has been involved in the inflammation. The accumulation of pus in the endolymphatic spaces is called endolabyrinthitis, the one in the perilymphatic spaces perilabyrinthitis. Suppurative paralabyrinthitis is suppurative inflammation of the osseous capsule of the labyrinth and petrous bone in the immediate vicinity.

Suppuration of the labyrinth is divided into diffuse and circumscribed, according to the way it has spread.

Suppuration of the labyrinth is diffuse if it extends to all the hollow spaces of the ear labyrinth, semicircular canals, vestibulum, and cochlea. If part of the spaces is exempt, the suppuration is called circumscribed.

Suppurative inflammation of the labyrinth is either acute, subacute, or chronic, according to the duration of the process.

In regard to the behavior of the neighboring parts, there are to be distinguished:

(1) Empyema of the labyrinth if there is accumulation of pus in the interior of the labyrinth but the surrounding bone is unchanged.

(2) Suppuration of the labyrinth with purulent paralabyrinthitis if the bone in the vicinity of the suppurative labyrinth has likewise undergone an inflammatory change.

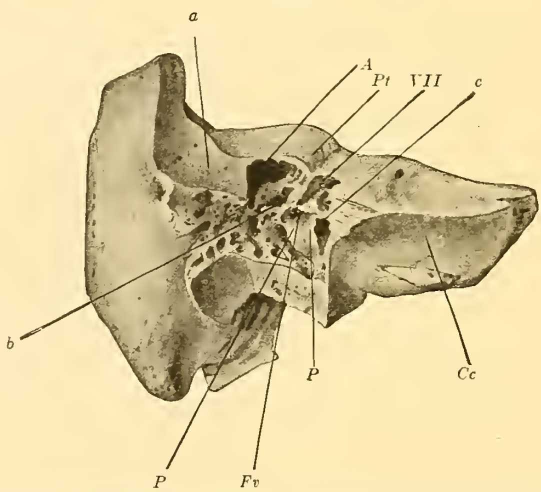
(3) Suppuration of the labyrinth with purulent paralabyrinthitis and fistula formation if there is suppuration of the soft labyrinth and a pathological communication has been established between the spaces of the labyrinth and the hollow spaces of the neighborhood.

Suppurative inflammation of the labyrinth is always infectious. Pathogenic micro-organisms can always be demonstrated by microscope as well as culture. Acute empyema of the labyrinth sets in with pronounced hyperæmia of the entire soft labyrinth. There is early coagulation of perilymph and endolymph, purulent decomposition of the coagulated masses, and destruction of the entire soft labyrinth. The canals through which the hollow spaces of the labyrinth normally communicate with their surroundings (aqueducts of the vestibulum and cochlea, the lymph fissures along the nerve canals of the petrous bone) are occluded

by fibrinous masses. The nerve-end places perish by cell necrosis as early as the first stages of the inflammation. The entire labyrinth permanently loses its function. The empyema, however, may persist unchanged for several months longer. A cure is gradually effected in favorable cases by resorption of the pus and by connective-tissue formation, which later changes into bone. In other cases the empyema will perforate outward into the middle ear or inward toward the endocranium into the middle or posterior cranial fossa. The perforation occurs by means of a fistula.

If the softening of the bone spreads from the fistular region to the rest of the petrous bone, the ultimate consequence will be caries or ne-

Fig. 93.



Suppurative paralabyrinthitis of the lateral wall of the labyrinth with carious destruction of the osseous wall of the auditory canal (*a*). Natural size. *A*, antrum mastoideum; *Pt*, paries tegminis; *VII*, canal of facialis; *c*, destruction of bone at the anterior portion of promontory and at the tympanic tubal ostium; *Cc*, canalis caroticus; *P*, promontory; *Fv*, fenestra vestibuli; *b*, destruction of lateral wall of the canalis facialis.

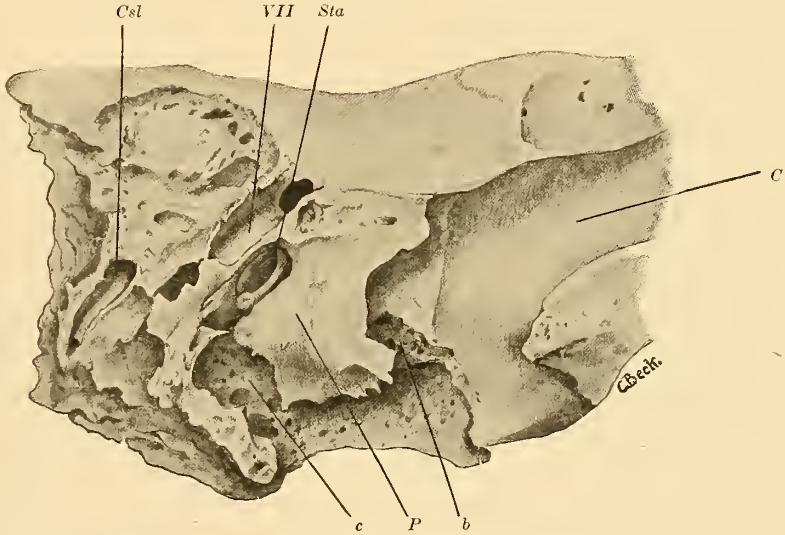
crosis of the labyrinthine nucleus of the petrous bone, or, less frequently, sequestration of the petrous bone itself, which is especially liable to happen in chronic tuberculosis of the labyrinth.

Suppuration in the neighborhood of the labyrinth will spread either by normal preformed anatomical routes or by pathologically formed canals. A suppurative process of the meninges may spread to the labyrinth by way of the internal auditory canal or the cochlear aqueduct, without causing any changes of the petrous bone. In all other cases of meningitic suppuration of the labyrinth and in all cases of otitic suppuration of the labyrinth, the direct spreading of the pus to the labyrinth occurs by way of fistulæ. Infection of the internal auditory canal occurs by way of the facial canal in those rare cases in which the pus accumulated in the middle ear can spread along the

nerve-sheaths of the facial by a congenital cleft of the osseous canal or a fistula.

Suppurative inflammation of the osseous capsule of the labyrinth (paralabyrinthitis) does not lead to any changes of the labyrinth spaces, as long as it does not reach the cortex which protects the spaces of the labyrinth. Should, however, the suppurative inflammation have advanced to the endosteum of the labyrinth spaces (perilabyrinthitis), there will be an inflammatory thickening of the endosteal layer of the perilymphatic tissue in the region of the fistula, followed by coagulation of the perilymph and endolymph, abundant quantities of mononuclear and polynuclear leucocytes in the lymph-spaces of the ear labyrinth,

Fig. 94.



Suppurative ostitis of the capsule of the labyrinth. Size 3.5: 1. *Csl*, fistula of the lateral semicircular canal; *VII*, canalis facialis destroyed by suppuration; *Sta*, stapes; *Cc*, canalis caroticus; *b*, suppurative focus in the canalis caroticus; *c*, suppurative focus in the hypotympanum; *P*, promontory.

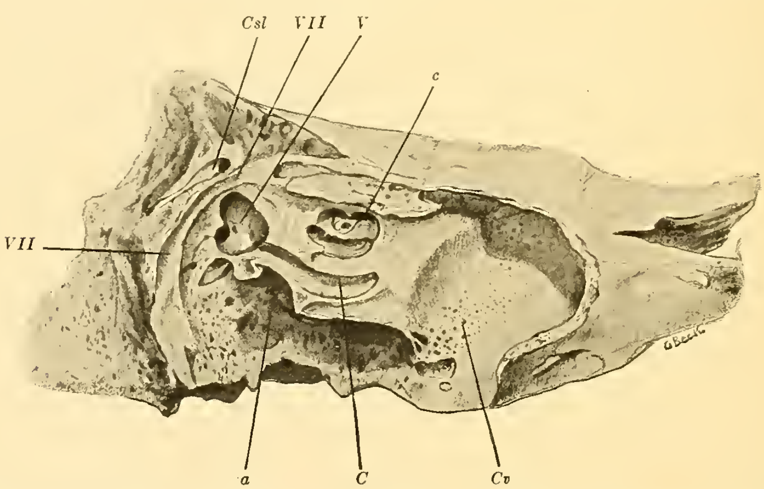
considerable thickening of the walls of the membranous labyrinth, and finally disintegration of the sensory epithelium at the nerve-end places. It is possible for the entire labyrinth to be involved in these changes or for the inflammatory manifestations to confine themselves to a part of the labyrinth. The process spreads chiefly by continuity of the perilymphatic spaces.

The labyrinth is divided into two compartments by the perilymphatic connective-tissue septum demarcating the cisterna perilymphatica vestibuli. One of these compartments contains the cochlea, the sacculus, and the cisterna vestibuli; the other one contains the semicircular canals, the ampullæ, and the recessus ellipticus. The resisting power of the connective-tissue septum, especially if it is thickened by

inflammatory infiltration, is sufficient to keep a circumscribed suppuration localized for some time in one of the two compartments of the labyrinth. Such a suppuration can even be confined for a short time to the lateral or posterosuperior semicircular canal. As a general rule, however, a circumscribed suppuration of the labyrinth will take place only in the acute or subacute stages, the type of the chronic suppuration being diffusely distributed over the entire labyrinth. Chronic circumscribed suppuration occurs only in those exceptional cases where a certain section of the labyrinth has been completely isolated from the other spaces by the formation of small sequestra or by a cholesteatoma.

A cholesteatoma of the labyrinth occurs from a middle-ear cholesteatoma, situated in the antrum or vestibular window, spreading to the

Fig. 95.



Caries of the petrous bone. All the spaces of the labyrinth are patent, owing to fistulæ. *Csl*, fistula of the lateral semicircular canal; *V*, fistula of the vestibulum; *C*, fistula of the cochlea; *Cc*, caries of the canalis caroticus; *a*, caries of the hypotympanum; *VII*, external wall of the canalis facialis is destroyed.

labyrinth. In this process there is usually extensive destruction of the petrous bone. Cases where the cholesteatoma spreads from a fistula to the labyrinth spaces without causing extensive bony changes are rare.

The thin osseous trabeculæ (crista vestibuli, scala, modiolus, lamina spiralis) are entirely destroyed by the chronic suppuration or the cholesteatoma of the labyrinth. That part of the petrous bone which is situated between the labyrinth and dura is likewise gradually destroyed. The final result is pachymeningitis externa, extradural abscess, and, finally, suppurative meningitis or cerebral abscess, with or without fistulous perforation into the dura. Suppuration of the labyrinth is often responsible for endocranial complications of the medial cranial fossa; should it occur, it almost invariably starts from a fistula or suppuration of the bone at the vertex of the upper semicircular canal. The

typical endocranial field of extension for a suppuration of the labyrinth is the posterior cranial fossa, more than 80 per cent. of the otitic cerebellar abscesses being etiologically traceable to suppuration of the labyrinth.

Symptomatology.—The following table will be useful in the study of the course of symptoms in diffuse, acute or chronic, suppurative labyrinthitis.

	Normal	I Initial stage	II Stage of inflammatory irritation	III Stage of inflammatory paralysis	IV Stage of healing	V Permanent findings after recovery
Spontaneous nystagmus	φ	Bilat'l	To affect. side	To the healthy side	Bilat'l	φ
Vertigo	φ	φ or +	+ or φ	+ or φ	φ	φ
Equilibrial disturbances	φ	φ	+	+	+	+
Reflex excitability of the static labyrinth	Normal	Normal	Pathologically increased	Pathologically reduced or negative	φ	φ
Hearing acuity	+	+	+	+ or φ	φ	φ

The division into five stages absolutely conforms to practical clinical experience. It should, of course, be considered that the transition from one stage into another is not necessarily distinct, as a given case may, for instance, present the symptom-complex of Stage I at one time and that of Stage II at another. Should vertigo occur in the beginning of the affection, the symptoms of Stage II will be found during the attack as well as a short time before and after,—namely, nystagmus toward the affected side and pathologically increased excitability of the labyrinth. The same patient, however, may present the symptom-complex of Stage I for a few weeks more when free from paroxysms: excitability of the labyrinth normal again and bilateral nystagmus.

The transition of Stage II into Stage III in suppuration of the labyrinth usually occurs with a violent paroxysm of rotatory vertigo. Deafness will set in simultaneously. If the hearing ability had previously been much reduced, the onset of deafness may escape the attention of the patient, especially if the hearing distance of the other ear is normal.

Nystagmus now occurs toward the non-affected side. The direction of the apparent rotation of the surroundings usually agrees with the direction of the nystagmus. While the patient previously perceived the apparent rotation of his surroundings toward the side of the affected ear, he now perceives it in the opposite direction. Patients rarely experience apparent rotation of their own body. During the violent paroxysm the nystagmus is very intense, rotatory, sometimes with a horizontal component. Usually, however, the intensity of the nystagmus is considerably relieved in a few hours, and in uncomplicated cases it is small and of slight intensity a few days later. The attack of vertigo is

sometimes of short duration, but in other cases it may last for several hours or days. The patient is unable to leave the bed, as the slightest movement, even the attempt to sit up in bed, brings on a violent accession of vertigo. Such an attack is also accompanied by subjective noises (tinnitus, ringing, etc.), nausea, and vomiting.

When labyrinthitis passes from the stage of inflammatory irritation into that of inflammatory paralysis, there is usually but one paroxysm of vertigo as the expression of the destruction of the nerve-end places (Stage III). In uncomplicated cases the vertigo attacks have now usually come to an end. Should, however, the excitability of the labyrinth not yet be completely destroyed, other less severe attacks may follow, which will cease after the membranous labyrinth has been destroyed by suppuration. After the attacks have ceased, there is still a slight sensation of continuous vertigo for a few days longer. This is gradually superseded by equilibrial disturbances, which, finally, likewise disappear. A careful examination is now necessary in order to discover to what extent the equilibrium has been disturbed by the destruction of the labyrinth.

Gradually, transition into Stage IV will take place. Spontaneous nystagmus toward the affected side is again demonstrable, but of less intensity. As time goes on, it may only occasionally be demonstrable or will completely disappear at the end of from six to eight months.

The course of uncomplicated suppuration of the labyrinth, therefore, extends over two to three months, counting from the beginning of the labyrinth symptoms and regarding the suppuration as terminated as soon as the spontaneous nystagmus has become very slight, and is directed toward both sides (to the right when looking to the right, and to the left when looking to the left).

1. ACUTE SUPPURATIVE PARALABYRINTHITIS WITH FISTULA FORMATION

It must be our endeavor to describe the various forms of inflammatory affections of the labyrinth as single clinical pathological pictures. In this way it is possible to take up separately the type of paralabyrinthitis with fistula formation, which deserves special clinical and anatomical interest.

Anatomy.—Acute paralabyrinthitis consists in suppurative inflammation of the lateral wall of the labyrinth and petrous bone in the region of the capsule. The suppurative inflammation of the bone extends up to the labyrinth. As a rule, erosion of the osseous capsule occurs in the area of the lateral semicircular canal, and, should the capsule be entirely destroyed, there will be diffuse suppuration of the labyrinth.

In cases which take a favorable course, there will be complete closure of the fistula, the function of the internal ear being retained.

It is uncertain whether the origin of the affection is favored by any special anatomical feature of the petrous bone. It would seem probable, however, that paralabyrinthitis has a better chance of development where there are extensive diploic or pneumatic spaces in the pyramid of the petrous bone than where the latter is composed of compact bone.

Occurrence, Symptoms, and Course.—Acute paralabyrinthitis with fistula formation more frequently follows in the wake of subacute than of chronic middle-ear suppurations, which extend to the antrum and mastoid process. It occurs exclusively in cases with repeated manifestations of pus retention in the middle ear and greatly reduced hearing ability during the whole course of the suppuration (conversation, 3 feet 4 inches). Participation of the antrum in the suppurative inflammation is indicated in these cases by early descent of the posterosuperior wall of the auditory canal. Spreading of the suppuration to the petrous bone and development of paralabyrinthitis are often favored by neglecting to open the mastoid process in time.

In these cases paralabyrinthitis may set in without any special symptoms, since headache, sensation of fulness in the ear, reduced hearing distance, and descent of the posterosuperior wall of the auditory canal may have existed previously. Slight spontaneous nystagmus (bilateral with lateral vision) is taken as an early symptom; sometimes there is also slight vertigo, which, however, is not characteristic. Usually, therefore, the alarming symptom of a sudden violent paroxysm of vertigo comes as a surprise.

Examination reveals violent nystagmus toward the affected side which occurs with vision toward the same side, often with straight vision or, in cases where the nystagmus is strongly pronounced, even with vision toward the opposite side. A distinct symptom of fistula can be produced in all these cases by pressing the tragus against the auditory meatus, which may cause a violent paroxysm of vertigo and a nystagmus-like slow movement of both bulbi toward the healthy side.

Diagnosis.—The diagnosis is not difficult. Paralabyrinthitis should be at once considered if in the course of a subacute middle-ear suppuration there occurs a paroxysm of vertigo due to the labyrinth. The characteristic diffusion of the disease is recognized by the positive fistular symptom.

Treatment.—Immediate antrotomy with broad incision of the antrum is indicated in paralabyrinthitis occurring in the course of an acute or subacute otitis. In subacute middle-ear suppuration these cases of paralabyrinthitis are not infrequently complicated by an extradural or mastoid abscess, from which descending abscesses will develop. The positive fistular symptom can be distinctly established during the

operation or during anæsthesia, a cotton tip advanced toward the antrum leading to a slow movement of the bulbi toward the healthy side.

Immediate radical operation is indicated where paralabyrinthitis has been caused by a chronic middle-ear suppuration.

Prognosis and course are favorable.

All the complaints disappear after the performance of antrotomy. The after-treatment does not differ from that in an ordinary antrotomy. Healing occurs in from six to eight weeks. The nystagmus toward the affected side disappears a short time after the operation; bilateral spontaneous nystagmus may persist for several weeks, but will gradually disappear. The fistula seems to close rapidly. In all these cases the normal function of the semicircular canals is perfectly retained, the hearing acuity rapidly improves, and patients will in most cases only experience a moderate reduction in their former hearing acuity.

The chronic cases may sometimes retain a satisfactory hearing acuity, but often there is a postoperative gradual decrease, until finally deafness sets in.

2. LABYRINTHITIS SEROSA

Etiology.—Labyrinthitis serosa occurs in rare cases as an independent affection of the ear, more frequently as a result of a traumatic injury to the head. It then assumes more or less completely the type of so-called traumatic concussion of the labyrinth. The occurrence of serous labyrinthitis as a complication of an affection of the middle ear seems to be intimately connected with the presence of a suppurative paralabyrinthitis which, up to the time, failed to present any symptoms. Under these circumstances it is possible for serous labyrinthitis to perforate through a trauma. Consequently it may occur in the course of acute as well as chronic suppuration of the middle ear. Serous labyrinthitis also includes the postoperative form which occurs in the wake of radical operations, after chronic suppuration of the middle ear has already induced superficial suppurative inflammation and erosion of the bone, which means that a superficial paralabyrinthitis has developed.

The symptoms of serous labyrinthitis consist in the sudden onset of vertigo due to the labyrinth, with simultaneous deterioration of the hearing acuity. There are also headache, lassitude, and moderate rise of temperature. The manifestations continue with unabated vigor for two or three days, during which there are repeated paroxysms of vertigo. At the climax of the disease there may be spontaneous nystagmus toward the non-affected side and transitory deafness. The virulence of the symptoms, however, will not last long. The nystagmus is presently directed again toward the affected side, then becomes bilateral,

and the hearing acuity re-establishes itself. All symptoms will have disappeared in about a week, and recovery takes place in nearly all cases with restitution of the reflex excitability of the static labyrinth. If the cochlea was intact previous to the onset of the affection, fairly good hearing will be re-established. Should, however, the cochlea and Corti's organ have undergone degenerative changes due to a preceding chronic suppuration of the middle ear, there is danger of the labyrinthitis being followed by permanent, insidiously developing deafness.

Treatment is purely symptomatic. During the violent irritative manifestations, rest in bed in a dark room, lying on the healthy side, is indicated. Usually a patient finds out himself in which position of the body and head he is least molested by vertigo and in which his nystagmus is least.

The diet should consist of small quantities of fluid, easily digested food. Violent attacks of vertigo may be relieved by the galvanic current (2–6 ma.), the current being conducted transversely through the head; 5 or 6 applications daily of from 2 to 5 minutes' duration. Sodium veronal (0.5–1.0), which has also been recommended in sea-sickness, may relieve the attack. In long-continued violent vertigo nothing but subcutaneous injection of morphine gives relief.

3. CIRCUMSCRIBED AND DIFFUSE UNCOMPLICATED (SIMPLE) SUPPURATION OF THE LABYRINTH

Etiology.—It may be said in a general way that only middle-ear suppuration which takes a grave course will lead to suppuration of the labyrinth. In acute or chronic middle-ear suppuration there is either otitis of the epitympanic type to deal with, or antrum suppuration with long-existing retention of pus, or severe middle-ear suppuration occurring in the course of scarlet fever or measles. In chronic cases there are usually neglected, highly fetid pus foci in the middle ear, which have long previously involved the bone or led to the formation of a cholesteatoma.

Symptoms.—There are frequent earaches and headaches. The pus secretion from the external auditory meatus varies, being sometimes abundant, sometimes slight, sometimes absent altogether. The labyrinth symptoms set in either without warning or after prodromal signs. The latter include headache, indistinct feeling of vertigo, lassitude, sensation of heaviness in the head or ear, involuntary twitching of the muscles supplied by the facial nerve on the affected side, or facial paralysis. Sometimes there are subjective noises in the form of a screeching, very high whistling sound. The typical sign of suppurative inflammation of the cochlea consists in a more or less sudden onset of deafness. If the patient has previously had fairly good hearing, the symptom of

deafness is distinct, especially if the affected ear has been the better one; but in chronic cases of middle-ear suppuration the hearing ability has already been bad before or been present only in remnants. The extinction of such remnants, which means the establishment of complete deafness, may entirely escape the patient's attention, especially when the other ear has normal or good hearing function.

In a few cases of chronic middle-ear suppuration, degenerative changes of the cochlea and Corti's organ may lead to deafness in the course of years. In any cases of this kind where deafness has occurred long ago, the onset of suppurative inflammation in the region of the cochlea will often occur without any symptoms.

The suppurative inflammation of the static labyrinth (vestibulum, semicircular canals) is associated with violent rotatory vertigo and equilibrial disturbances. Should there be a sudden diffuse suppuration of the labyrinth with a hyperacute course, in which the entire sensory epithelium is immediately destroyed, there will be only one, but very violent attack of rotatory vertigo. The attack is always described correctly by the patient, and, owing to the violence of the attack, he will usually be able to indicate the direction in which the rotation occurred. The rotation is usually passive,—a rotation of the surroundings; less often an apparent rotation of the patient's own body. (An apparent rotation of the surrounding objects to the right corresponds to an apparent rotation of the patient's own body to the left.)

The attack of vertigo is associated with violent nystagmus due to the labyrinth. The direction of the apparent rotation of the surrounding objects usually corresponds to the direction of the nystagmus. If, therefore, a patient states having had the perception of objects around him turning to the right, it is always equivalent to the presence of a right nystagmus, being directed toward the same side. The sensation of an apparent rotation of the patient's own body to the right, however, is usually associated with the presence of a labyrinth nystagmus toward the left.

Nystagmus and vertigo are often present during the early stage of a suppuration of the labyrinth. The nystagmus is not intense and, therefore, only demonstrable with lateral vision. At the same time spontaneous bilateral nystagmus toward the affected side may be observed, and, besides, the kind of nystagmus may alternate. If there is vertigo, there will be spontaneous nystagmus toward the affected side, while during the periods when there is no vertigo there is either bilateral nystagmus or none at all.

The increase of vertigo which indicates the onset of diffuse suppuration of the labyrinth is always accompanied by violent spontaneous nystagmus of the greatest possible intensity toward the healthy side.

It is most pronounced with vision toward the side of the nystagmus, which means toward the healthy side, but it will also persist with straight vision, even with vision toward the affected side. The patient exhibits the signs of a marked subjective or objective rotatory vertigo. If the paroxysm seizes him at home, he will instantly go to bed, or lie down prone on the floor, remaining in this position until somebody comes to his aid to put him to bed. If the paroxysm seizes him in the street, he is exposed to the most severe accidents.

A few years ago a patient was taken to the hospital by the First-aid Society, with the following report: The patient suffered from chronic suppuration of the middle ear, and had a few weeks previously complained of periodical, unimportant vertigo. On the day of admission he went to work in the morning, and in the street was overcome by a violent paroxysm. He was only just able to clutch a lamp-post, to which he held spastically until he was rescued by an officer of the First-aid Society and placed on a stretcher.

Concomitant manifestations of such a violent paroxysm are equilibrial disturbances and repeated vomiting. The equilibrial disturbances occur in conjunction with the signs of objective vertigo, and find expression in considerable staggering, positive Romberg's symptom, inability to stand on one leg with closed eyes, and in disturbances when walking forward or backward with closed eyes. Should there be a very high degree of vertigo, there can be no question of standing or walking; the patient may even be unable to sit up in bed. While in bed he will instinctively find the best position for body and head, in which the nystagmus is least. In that position he will feel the vertigo less than in any other. It will invariably be observed that patients with suppuration of the labyrinth and nystagmus toward the healthy side lie on the affected side, or at least turn the head in that direction. Having at last found the most comfortable position, the patient will be compelled to relinquish it frequently by repeated vomiting. During an attack lasting for two or three hours, the patient may have to vomit 20 or 30 times; in any case there is always nausea during the entire duration of the attack, the patient being unable to retain even the slightest quantities of fluid.

The acute stage of the first attack is followed by a more or less continuous sensation of vertigo. Without being quite free from giddiness, its intensity is considerably diminished, vomiting has ceased, and the sensation of rotation now exists only with vision toward the healthy side, as long as the nystagmus prevails. He can retain food, after a few days he can sit up in bed without being particularly bothered by vertigo, and after from 8 to 14 days the latter has completely disappeared. Examination of the internal ear will then reveal total deafness and absence of excitability of the vestibulum and the semicircular canals. If the sensory

epithelium has not been completely destroyed at the beginning of the suppuration of the labyrinth, some hearing remnants and reflex excitability of the static labyrinth will, at first, still be retained. In these cases there is nystagmus toward the affected side at the first attack of vertigo. These cases, which belong to the class of circumscribed suppurations of the labyrinth, sometimes recover at this stage of the affection, retaining at first some hearing remnants and excitability of the semicircular canals. Should the suppuration make further headway, there will be renewed accessions of vertigo, the last of which will be characterized by nystagmus toward the healthy side and terminate with complete destruction of the labyrinth, which means deafness and absence of excitability of the semicircular canals.

Uncomplicated suppuration of the labyrinth is not accompanied by any local cerebral symptoms. The fundus of the eye and the cerebrospinal fluid are normal. The temperature in acute suppuration of the labyrinth is moderately raised. There are no unilateral disturbances of coördination. The reflexes, superficial and deep sensitiveness are normal.

Diagnosis.—The diagnosis of suppuration of the labyrinth is easily made when in a case of middle-ear suppuration or suppurative meningitis there are accessions of rotatory vertigo and sudden deafness. The diagnosis becomes more difficult the less abrupt the labyrinth symptoms occurred and the longer the symptoms have existed without leading to complete loss of function of the internal ear.

In circumscribed suppuration of the labyrinth the history will reveal attacks of vertigo, while examination will show that part of the labyrinth is still functioning and the rest has lost its function. There may, for instance, be positive hearing, although the excitability of the semicircular canals is extinct and there is permanent diminution of the equilibrium. The diagnosis is more difficult in cases of circumscribed suppuration of the labyrinth, where nothing but the acoustic apparatus has been destroyed while the semicircular canals have retained their function. The diagnosis can then be made if the history establishes the fact that deafness has occurred in an apoplectiform manner.

As to diffuse, uncomplicated suppuration of the labyrinth, there can be no difficulty in making the diagnosis with attentive observation of the patient and sufficient experience in the interpretation of spontaneous nystagmus, although the preceding attacks of vertigo which have been established by the history do occur in other affections of the labyrinth as well. Of particular value from an anamnestic and diagnostic point of view, however, is the isolated violent attack of vertigo, under which Stage II has developed into Stage III, together with the occurrence of deafness.

As to the differential diagnosis, the following affections have to be

considered: (1) serous labyrinthitis; (2) hemorrhage of the labyrinth; (3) acute serous inflammations of the labyrinth (neurolabyrinthitis) and acute degenerations of the labyrinth in cases of typhoid and syphilis; (4) traumatic injury to the labyrinth (fracture); (5) neurasthenia of the labyrinth.

Differentiation between diffuse suppuration and serous labyrinthitis is rendered possible by the fact that in the latter the stage of inflammatory paralysis is not reached at all or only for a short time and, instead of Stage IV following, there is return to Stage II and later to Stage I, with restitution of the excitability according to the course of the healing process of the serous labyrinth.

Apoplectiform non-traumatic hemorrhages of the labyrinth are nearly always associated with lymphomatous affections of the blood. The labyrinth, which previously was normal, is completely destroyed by an effusion of blood. The differential diagnosis can at once be established by the blood findings of leukæmia, and, besides, there are slight attacks of vertigo in diffuse suppuration of the labyrinth preceding the violent paroxysm, whereas in cases of hemorrhage of the labyrinth there have never been any such attacks before. The paroxysm in hemorrhage of the labyrinth is exceedingly violent, but of considerably shorter duration than in suppuration of the labyrinth. Furthermore, the chronic vertigo which follows the acute attack in suppuration of the labyrinth is more or less absent in hemorrhage of the labyrinth.

In syphilis of the labyrinth or acute degeneration in typhoid the isolated violent paroxysm of vertigo is preceded by a very large number of lighter attacks.

The differentiation from trauma of the labyrinth can be made from the history regarding the trauma itself. This is supported by symptoms of other cerebral nerves (trochlearis, abducens, facialis). Besides, it is important to remember for the diagnosis of suppuration of the labyrinth that an existing previous suppuration of the middle ear or an intracranial suppuration must be established.

Treatment.—Many uncomplicated cases of empyema of the labyrinth recover spontaneously. Rest in bed is urgently needed from the onset of the affection, and may have to be continued for many weeks, until the symptoms have completely subsided. During the period of violent attacks it is advisable to darken the room and to bring patient into a position where his nystagmus is felt least. Most patients instinctively find that position themselves.

The diet must be selected with the utmost care. Small quantities of fluids are administered by the spoonful, without changing the patient's position if possible. Stimulating beverages, such as alcohol, are rigorously excluded.

As to galvanization, etc., see p. 265. Subcutaneous morphine injections may be necessary in the violent attacks.

Diffuse suppuration of the labyrinth which has originated from meningitis may recover spontaneously under this treatment; the otitic form, however, will only so recover if the osseous capsule of the labyrinth has remained intact. Should the osseous region between the membranous labyrinth and the middle ear be affected, resection of the labyrinth will be necessary.

4. COMPLICATED DIFFUSE SUPPURATION OF THE LABYRINTH

Complicated diffuse suppuration of the labyrinth is the result of the inflammation involving the anatomical region of the labyrinth either by direct spreading or by metastasis. Accordingly, there are to be distinguished endocranial and extracranial diffuse suppurations of the labyrinth.

(a) *Suppuration of the Labyrinth Complicated by Extracranial Involvement*

This group comprises the diffuse suppurations of the labyrinth in the course of which suppurative osteitis of the petrous bone, fistula formation at the lateral wall of the labyrinth with sequestration of that wall, or paralysis of the facial nerve have developed.

Anatomy.—In diffuse suppuration of the labyrinth the involvement of the lateral wall is usually a deuteropathic manifestation or the termination of a labyrinth suppuration. Under the influence of chronic middle-ear suppuration, there will be sequestration of flat, minute particles of the lateral labyrinth wall, particularly at the promontory; more frequently, however, carious destruction and fistula formation consequent upon pus breaking through from the labyrinth toward the middle ear. The fistula either develops over preformed anatomical tracts (vestibular and cochlear windows), or else the bone will be destroyed at those places where the capsule of the labyrinth is normally thin (eminence of the lateral semicircular canal, promontory). In the presence of multiple fistulæ the entire wall of the labyrinth may be sequestered. This is followed by exfoliation of the osseous parts situated in the labyrinth itself (modiolus, lamina spiralis, crista vestibuli); in the end, sequestration of the entire petrous bone may occur, especially in tuberculous cases.

Should the suppurative inflammation of the bone reach the facial nerve, there will be suppurative infiltration of the nerve-sheaths and finally of the nerve itself. A cholesteatoma of the labyrinth, if present, may completely destroy the facial nerve, enabling the pus to spread either along the facial canal into the endocranium or peripherally toward the base of the petrous bone.

Symptoms.—Suppurative osteitis may exist for a long time without giving rise to any symptoms if the entire labyrinth has been destroyed by diffuse suppuration.

An objective sign of the affection of the lateral wall of the labyrinth capsule consists in the otoscopic demonstration of sequestered bones, in the spontaneous expulsion of minute sequestra, and in the demonstration of labyrinth fistulæ. The pus of the middle ear is fetid in the highest degree; the quantity of the secretion varies, and there may be no secretion at all for days together.

The otoscopic demonstration of a fistula or sequestrum is possible only in exceptional cases, while the expulsion of sequestra is more frequently observed. Patients sometimes preserve these, and, upon careful examination, they are found to be parts of the capsule or interior of the labyrinth. Labyrinthogenic paralysis of the facial nerve is a positive but often very unfavorable symptom. The paralysis sets in insidiously. Prodromal signs in the shape of involuntary twitching of the mimic musculature and transient paralysis of some group of muscles may last for one or two weeks, and it is only then that permanent paralyses will set in and continue to develop until, in the end, the picture of a complete peripheral unilateral paralysis presents itself. This is explained by the fact that the suppuration spreads to the facial nerve in a gradual way, involving first the peripheral bundles of the nerve and later the fibres which are axially situated in the trunk. Temperature is usually somewhat raised.

Course and Treatment.—Spontaneous recovery without operation may occur in exceptional cases; otherwise endocranial labyrinth fistulæ, meningitis, cerebellar abscess, or caries of the petrous bone will develop, while the fetid suppuration continues. Consequently, nothing but operation will afford radical treatment, and this consists in the exposure of the middle-ear spaces and resection of the labyrinth. The latter has to be preceded by exposure of the dura of the middle and superior cranial fossæ.

Labyrinth Operation.—Complete evacuation of the mastoid process is a necessary preliminary step. Then the dura of the posterior and medial cranial fossæ is dissected free, and the two apertures thus obtained are united by removing the superior edge of the pyramid. The dura having been carefully mobilized, the labyrinth is exposed step by step in the direction of the affected bone. Commencing at the semicircular canals, the petrous bone is removed with the chisel, starting from the exposed posterior fossa.

Spontaneous evacuation of the cerebrospinal fluid from the labyrinth spaces is a sure sign of the affected parts having been sufficiently drained. This evacuation can be attained in all acute cases. In older

cases it may be rendered impossible by the accumulation of blood coagula in the labyrinth spaces, by pathological connective tissue or new-formed bone. A cholesteatoma may likewise prevent the evacuation after resection of the labyrinth. In these cases the vestibulum has to be opened after removing the semicircular canals. In all cases, however, the cochlea must be opened by cutting away the promontory. If labyrinth fluid flows out of the cochlea, the operation may be stopped. Should the cochlea, however, contain pus or a cholesteatoma, it will have to be emptied with a sharp spoon.

The wound is loosely tamponed with iodoform or isoform wicks; one bundle of wicks is conducted to the cochlea and outward through the external auditory meatus, and a second bundle toward the vestibulum or the semicircular canals (which have been opened) and conducted outward through the retro-auricular wound. The latter is left open and should only be closed by suture at the end of the first week if the healing process takes a favorable course.

The prognosis of diffuse suppuration of the labyrinth without intracranial involvement is not unfavorable if the operation has been done in proper time. Recovery will usually take much longer than that of an ordinary radical operation. It occurs under formation of a resistant layer of cicatricial tissue which completely covers the petrous bone, leaving a spheric cavity, invested with glistening gray epidermis.

The prognosis of paralysis of the facial nerve is dependent upon the electric excitability of the nerve and on the local changes found at operation (see chapter on Paralysis of the Facialis.)

The subjective condition of the operated patients is good in most cases, but they should abstain from any strenuous work even after complete recovery. There remains a tendency to rapid fatigue for months afterward, and headache will easily occur upon great mental or physical exertion. Neurotic patients may experience neurasthenic complaints even after an ideal healing process, which may simulate an endocranial complication or the continuance of an inflammatory affection of the labyrinth.

(b) *Diffuse Suppuration of the Labyrinth Complicated by Endocranial Involvement*

Under this head all those cases are summarized in which a diffuse suppuration of the labyrinth has led to a suppurative inflammation of the endocranium either by direct spreading or by metastasis.

Anatomy.—Direct spreading of the pus from the labyrinth to the endocranium leads to destruction of that part of the labyrinth capsule which is situated between the labyrinth and the cranial fossæ, to destruction of the petrous bone, and to the formation of one or more endo-

cranial fistulæ. The fistula either takes its way through preformed canals (internal auditory canal, cochlear or vestibular aqueduct, superior semicircular canal, fossa subarcuata), or it develops, like an extracranial fistula, where the osseous layer between the spaces of the labyrinth and the dura is particularly thin (superior semicircular canal, commissure of the semicircular canals, sinus half of the posterior semicircular canal). Involvement of the posterior cranial fossa is far more frequent in endocranial complications than involvement of the middle cranial fossa.

Should the suppuration spread through the internal auditory canal over the cochlear aqueduct, pachyleptomeningitis will develop. In all other cases there will at first occur a pachymeningitis externa, in the wake of which an extradural or cerebellar abscess will develop.

Spreading of the suppuration over the vestibular aqueduct and the endolymphatic duct may result in an extradural abscess at the external aperture of the vestibular aqueduct or a saccus empyema.

Metastatic spreading of the labyrinth suppuration may lead to suppurative meningitis or cerebellar abscess with or without involvement of the interpolated layers.

Suppurative sinus phlebitis is only rarely caused by suppuration of the labyrinth; at least it is only exceptionally possible to prove that in these cases the sinus thrombosis has been caused by the suppuration of the labyrinth and not by the underlying cause, the chronic middle-ear suppuration. Thus, fistulæ of the upper semicircular canal may lead to infection of the sinus petrosus superior and thence to suppurative phlebitis and pyæmia. Saccus empyema and labyrinthogenic extradural abscesses of the hindmost region of the posterior cranial fossa may be followed by suppurative inflammation and thrombosis of the sigmoid sinus.

Symptoms and Course.—Tormenting headache usually occurs without any warning as soon as the suppuration of the labyrinth has reached the dura. The headache usually occurs in the vertical region if the dura of the middle cranial fossa has been affected; the pain will be at the occiput or base of the skull if the dura is affected at the posterior surface of the petrous bone. In the latter case patients will avoid any sudden movement of the head. Active movements of the head are in many cases considerably restricted, while, according to the most careful investigations, the passive movements are fully maintained. Accordingly, there is moderate stiffness of the neck and often abnormal inclination and torsion of the head toward the affected side.

At the time that diffuse suppuration of the labyrinth is in active operation, irritative manifestations of the labyrinth are usually no longer present, and their former occurrence can therefore only be established by the history. Vertigo, equilibrial disturbances of any importance,

and vomiting now occur only on mechanical irritation or by suppurative inflammation of the eighth nerve in cases where the suppuration has spread to the endocranium along the nerve-bundle of the internal auditory canal. In these cases it is even possible for a new positive fistular symptom to be elicited from time to time if any extracranial fistulæ exist. Examination of the ocular fundus often reveals abnormal venous plethora and sometimes optic neuritis at the affected side. Spreading of the suppurative process to the pyramid of the petrous bone will be followed by abducens paresis, or complete abducens paralysis with double vision. Temperature is moderately raised; usually there is anorexia and sometimes vomiting. There are depression, dislike of work, and a desire to rest in bed. Lumbar puncture, even in the early stage, yields a turbid fluid in many of these cases. The evacuated fluid will, on standing, form slight coagula, which microscopically show abundant mono- and polynuclear leucocytes, and often micro-organisms (staphylococci, streptococci, etc.). In many of these cases where the findings as to micro-organisms are positive, cultures and animal tests will prove negative, but change to positive as the affection makes further progress. Should the suppuration lead to the formation of a cerebellar abscess, the corresponding symptoms will now make their appearance. A detailed description of the same is contained in the section on Cerebellar Abscesses.

Diagnosis.—Should headache, rise of temperature, or impaired movements of the head persist, after the paroxysms of vertigo and the manifestations of intense labyrinth nystagmus have abated, it is safe to assume an endocranial involvement induced by the labyrinth.

The diagnosis is more difficult when at the time of endocranial involvement the manifestations on the part of the labyrinth have not yet disappeared, particularly when the patient still complains of vertigo.

A differentiation is impossible if the suppuration has spread to the eighth nerve in the internal auditory canal, as the inflammatory processes of the formerly non-affected nerve may cause similar manifestations as those of a fresh suppuration of the labyrinth. In such cases the diagnosis may be rendered possible by the complaints continuing after resection of the labyrinth or their recommencing after a slight interval. However, by strict adherence to the rule to precede every resection of the labyrinth by exposure of the dura, the endocranial complication will be determined at the operation, should it not have been possible to recognize it clinically.

Treatment.—Early operation offers the only chance of recovery. This consists in opening the middle-ear spaces, in the free exposure of the posterior and medial cranial fossæ, removal of the superior edge of the petrous bone, and resection of the ear labyrinth. In the presence of suppurative inflammatory changes of the nerve-trunk, it is necessary to expose the internal auditory canal by removal of the lateral osseous wall

which is situated between the vestibulum and the auditory canal. After this the dural lining of the internal auditory canal is incised. This form of resection, together with removing the cochlea, will expose the intradural space of the posterior cranial fossa at the internal auditory canal. It now communicates with the middle-ear spaces through the internal auditory canal and the cochlear aqueduct. It is advisable to incise the dura of the posterior cranial fossa in the region of the resection, because it is just these small incisions which invite the danger of direct migration of pathogenic germs into the cranial cavity.

Should the cerebellum be unchanged, there will be no prolapse of the bone. In the presence of encephalitis, the œdematous cerebellum will protrude into the incision. In these cases there may be persistent cerebellar prolapse, which may later be remedied by skin plastic operations. Sometimes the prolapsed portion becomes necrotic and sloughs off, with consequent healing, but there may also occur an intrameningeal abscess in the shape of large accumulations of pus at the fundus of the posterior cranial fossa.

In such cases the sudden onset of a fulminating, diffuse suppurative meningitis, many weeks after the operation, may lead to death within a few days, in spite of the apparently favorable course.

In cases which take a favorable course, the healing process does not differ from that in ordinary resections of the labyrinth. The occasional expulsion of small sequestra will not permanently disturb the healing process.

A compilation of the cases of suppuration of the labyrinth with endocranial complications which have been operated upon in my department shows a mortality of about 20 per cent., including the cases of labyrinthogenic cerebellar abscesses. The prognosis of labyrinthogenic suppurative pachyleptomeningitis, when restricted to the posterior cranial fossa, is relatively favorable, although the fact may seem remarkable.

XII. EXTRACRANIAL AFFECTIONS OF THE EAR

Extracranial affections of the ear occur by a middle-ear suppuration leaving the region of the middle ear and advancing into the surrounding territory except toward the cranial cavity.

Accordingly, we distinguish:

(1) Subperiosteal mastoid abscess.

(2) Perforating abscess toward the squama of the temporal bone and the zygomatic process.

(3) Perforation to the external auditory canal from the fistulæ of that canal.

(4) Descent of pus toward the submaxillary bone, sometimes with periarticular suppuration into the submaxillary articulation.

(5) Descent of pus along the tube.

(6) Descending abscesses of the neck: (a) perforation of mastoid abscess through the pyramid, (b) through the middle wall of the mastoid.

It is in the nature of the anatomical structure of the temporal bone that all the variations above enumerated result in most cases from the direct extension of spreading of pus. There is, consequently, an immediate anatomical communication between the pus focus of the middle ear and the extracranial focus. The smaller part of extracranial complications are caused by metastatic suppuration or by phlegmonous inflammation originating in the ear.

I. SUBPERIOSTEAL MASTOID ABSCESS

A subperiosteal mastoid abscess is occasioned by an accumulation of pus between the periosteum and the lateral wall of the mastoid process. It occurs in the course of a suppurative inflammation of the mastoid process if the abscess in the bone finally perforates outward through an osseous fistula underneath the periosteum. The fistula is usually situated in the mastoid fossa, near the antrum in the upper part of the mastoid process. In fistulæ of the posterosuperior wall of the auditory canal the pus may gradually lift up the soft parts of the osseous auditory duct, force a way underneath the periosteum, and finally collect in an abscess above the mastoid process. Metastatic subperiosteal mastoid abscesses without any mastoid fistulæ are rare in infancy and childhood, and the same is true of subperiosteal mastoid abscesses in simple suppuration of the middle ear without any distinct clinical sign of the mastoid being involved in the inflammation. In such cases the metastasis occurs through the small veins of the soft covers of the mastoid which extend into the middle ear. Fistulæ which are situated below the level of the

insertion of the sternocleidomastoid muscle lead to descending abscesses of the neck, and no longer to periosteal abscesses (Figs. 101, 102).

Subperiosteal mastoid abscesses occur oftener in acute suppurative mastoiditis than in the chronic form. They occur early in a diploic abscess when the cortex is thin. Their occurrence is particularly rapid in mastoiditis of children under four years of age. Here the bone is exceedingly vascular, the cortical layer thin, the mastoid largely or entirely diploic. Besides, the infantile lateral wall of the antrum is thin and often still cartilaginous in parts. In rhachitic bony changes, which are of frequent occurrence, the resistance of the cortical layer to the invading pus is still more reduced and the bone is rapidly destroyed. Under these circumstances it is not surprising that, as early as a few hours after the occurrence of mastoid manifestations, in infancy, a subperiosteal perforation and fluctuation have occurred. Where the cortical layer is thick, however, perforation will not occur before the lapse of four or five weeks.

Subperiosteal abscess is rare in chronic middle-ear inflammation. This is explained by the fact that in the course of the latter there occurs a gradual thickening and partial sclerosis of the osseous structure of the mastoid process without any acute inflammatory manifestations. This will in most cases effectually check a spreading of the suppuration to the mastoid and prevent a perforation through the lateral surface of the same. To allow the suppuration to spread to the mastoid, a fresh acute attack of the chronic suppuration or an acute suppurative decomposition of a cholesteatoma of the middle ear is at least required. The latter is the more frequent occurrence.

In these cases, however, the subperiosteal abscess is rarely the only otitic involvement. More frequently it is merely a part manifestation of an endocranial complication caused by the rekindling of a chronic suppuration or by the acute suppurative decomposition of a cholesteatoma. In subacute purulent mastoiditis a subperiosteal mastoid abscess is often the sign of an impending mobilization of a sequestrum.

Anatomy.—It occurs only in cases of tuberculous subperiosteal abscess that there is an accumulation of pus within closed walls without reaction. In all other cases the middle wall of the abscess,—*i.e.*, the lateral wall of the mastoid,—as well as the lateral wall of the abscess, presents more or less important inflammatory changes. The fistulæ found in the cortical layer have already been mentioned. They are sometimes canals with such narrow lumina as to be passable only by a small sound or a blunt needle. They may, however, attain the size of a goose-quill, ending in a large aperture, which gives at first sight the appearance of total destruction of the cortical layer of the mastoid, of a broad communication between the endomastoid and subperiosteal pus

foci, and a confluence of both abscesses into one. Sometimes there are several fistulæ, which may cause a cribriform perforation of the bone in the presence of a thin but resistant cortical layer. The fistula is gradually filled with granulations as the abscess continues its existence, while the lateral surface of the mastoid process is relatively seldom the seat of granulations. The soft layers over the abscess are usually thickened by calosities which may attain a thickness of ½ to 1 cm. in advanced cases. These growths generally occur by fibrinous deposits between the periosteal layers as well as between the periosteum and the subcutaneous cell tissue. The fibrin may become organized, leading to sclerosis of the connective tissue. The periosteal wall facing the abscess is usually covered with granulations. Its injected and strongly osmotic condition sharply demarcates it against the periosteum of the normal surroundings.

As soon as the abscess is about to perforate, there occur infiltration, reddening, and swelling of the skin; finally the skin assumes a livid hue in one or more places, and by destruction of a circumscribed area a retro-auricular fistula will be formed, through which the abscess pus and that of the mastoid process are evacuated. The skin fistula is in most cases situated on the level of the osseous fistula of the mastoid process, so that a probe introduced into the skin fistula will penetrate into the interior of the mastoid without meeting with any resistance.

The size of subperiosteal abscesses varies from a pea to a man's hand. This extraordinary enlargement is occasioned by the pus gradually lifting the periosteum beyond the area of the temporal bone. It usually penetrates first the squama of the temporal bone, then the occipital bone, the zygomatic process, and finally the parietal bone. Spreading beyond the vertex to the parietal bone of the other side occurs only in rare cases.

I have observed three cases where the fluctuation was demonstrable from the central part of the mastoid region backward to the nape, thence forward to the zygomatic process, and upward to the parietal bone of the other side, thus crossing the vertex.

Fetid pus is found only in cases of chronic middle-ear suppuration, and is usually a part manifestation of an intracranial complication, the most frequent of which is a sinus thrombosis.

Subperiosteal gas abscesses are very rare and occur only in cases of chronic middle-ear suppuration with endocranial complications.

Symptoms.—The local findings are furnished by the abscess itself. In the mastoid region there is a swelling in the shape of a flattened globe, the skin of which is drawn tight over it and often becomes hyperæmic and lividly discolored upon impending perforation. The more or less perceptible fluctuation depends upon the size of the abscess and upon the extent to which the subcutaneous cell tissue has been destroyed. Spontaneous tenderness of the mastoid is rare in these cases; it may be en-

tirely absent, and principally depends upon the tension of the abscess wall. Abscesses with scant contents do not cause spontaneous pain, but there is a more or less pronounced degree of pain on pressure. The purulent contents can be easily seen by transillumination and comparison with the other side. The concha stands away from the head, sits lower than its mate, and has a forward and downward torsion (Fig. 96).

The tympanic changes agree in most cases with those in acute mastoiditis. Sometimes, however, the local inflammatory manifestations have entirely subsided, so that both tympanic membrane and hearing acuity are quite normal. Testing, however, the bone-conduction over the abscess, considerable reduction will be found, which is due to the fact that the abscess (fluids and bad sound conductors) is interposed between the foot of the tuning-fork and the osseous surface. The temperature of the patient is moderately elevated. There are neither cerebral symptoms nor general manifestations. Often there are complaints

Fig. 96.

Pathognomonic change of position of the right concha in right-sided subperiosteal mastoid abscess. The change of position is best observed in the dorsal aspect.

of tormenting, knocking, subjective noises and pulsation in the region of the affected ear. Sometimes there is a widely diffused œdema of the face, with occlusion of the lid fissure and swelling of the cheek. In these cases mastication is likewise impaired.

Diagnosis.—There can be no diagnostic difficulty in view of the objective abscess symptoms. The history, otoscopic findings, and the characteristic position of the concha distinctly betray the otitic character of the abscess.

In the presence of a subperiosteal mastoid abscess it is of the greatest importance not to overlook any intracranial otitic affection that may be present. It has already been stated that subperiosteal abscesses in chronic middle-ear suppuration nearly always represent a part manifestation of an endocranial complication.

In acute cases the above symptoms will be all to go by, remembering, however, that a subperiosteal mastoid abscess is not accompanied by

serious local or general manifestations. Should such be present, therefore, —such as headache, delirium, convulsions, high temperature, intermittent fever, chills, icterus,—we must not be content with the diagnosis of subperiosteal mastoid abscess, but must look for an endocranial affection.

In making a *differential diagnosis* subperiosteal abscesses of non-otogenic origin, which have spread secondarily to the mastoid region, should be considered first. These are nearly always cases of tuberculous osteoperiostitis of the zygomatic process or squama of the temporal bone, in very rare cases of the occipital bone. The history is to be taken as the first guide (absence of a previously existing middle-ear inflammation), also the development of the disease (no middle-ear symptoms, fever, or pain). A careful examination of the patient, and especially the operative findings, will permit of determining which was the bone primarily affected.

Glandular abscesses and bundles situated at the musculus splenius capitis may simulate mastoid abscesses, especially if the patient has also middle-ear symptoms or the history reports such symptoms. Examination will generally, however, show that the abscess does not extend beyond the posterior half of the mastoid process and will not reach to the frontal part, but terminate sharply demarcated at the mastoid process itself. The concha is in normal position, the mastoid pyramid is distinctly palpable, and the external auditory canal is unchanged. In doubtful cases the decision must depend upon the operative findings. The middle wall of a subperiosteal abscess is always formed by exposed bone, while the middle wall of a glandular abscess is covered with callous connective tissue or remnants of the glandular covers, and the periosteum is intact.

Furunculosis of the auditory canal may in rare cases lead to descent toward the mastoid process, even to abscess formation. The diagnosis in these cases is easy, and, besides, the operative findings (periosteum unchanged, pus between the skin and periosteum, no subperiosteal pus) will guard against error. In tuberculous perichondritis descent toward the mastoid process may be caused by extensive cartilaginous necrosis. The abscess is situated on the level of the apex of the mastoid and usually extends forward to the middle surface of the lobulus. The abscess shows all the signs of a tuberculous ulceration.

The differential diagnosis has still to take into consideration subperiosteal hæmatoma in fractures of the base of the skull which pass through the mastoid process. History and transillumination (dark-red diaphany) will enable us to recognize the hæmatoma as such. In some cases, however, suppuration of the hæmatoma may develop later. Should there be grave cerebral manifestations aside from the fracture, a clinical differentiation will be impossible, but at operation, after evacuation of

the abscess and after exposure of the mastoid process, the fissure of the fracture, filled with blood and pus, will be recognized.

The diagnosis of actinomycotic abscesses will be made from the microscopic findings of the fungi.

Treatment.—Œdematous swelling of the soft covers of the mastoid often recedes spontaneously or under conservative treatment, especially in children. Generally speaking, however, the treatment of subperiosteal mastoid abscess can only be surgical. The lateral surface of the mastoid process is exposed through the typical retro-auricular incision. In larger abscesses the greater portion of the contents may be evacuated with a trocar. If the outward perforation has already set in, the skin incision is made through the fistula, and the fistular wall is removed with the scissors. The abscess wall is cleansed with sharp spoons and sharp curettes, after which the mastoid process is opened in the typical manner.

In acute middle-ear suppuration simple opening of the mastoid (mastoidotomy) will suffice, provided the bone in the direction toward the antrum is healthy. This holds especially good for all those cases where a suppurative middle-ear inflammation in the region of the tympanic membrane has already run its course at the time of the operation (tympanic membrane closed, good hearing acuity). Should there be still secretion from the external meatus, antrotomy is absolutely necessary. No cure may be expected from a mere puncture of the abscess or a simple incision.

In infants antrotomy should be done in all acute cases.

In chronic suppuration of the middle ear, radical operation has to be resorted to, as a matter of course. The extent of the same is to be adapted to the requirements of the middle-ear changes.

Prognosis.—The prognosis and the postoperative course depend upon the underlying affection.

In large abscesses which have existed for a long time and led to extensive destruction of the periosteum, large, deep scars are to be expected.

II. OSTEOPERIOSTITIS AND SUBPERIOSTEAL ABSCESSES OF THE TEMPORAL SQUAMA AND THE ZYGOMATIC PROCESS

Subperiosteal abscesses in the region of the temporal squama and the zygomatic process are rather rare. They occur by outward perforation of the pus from the middle ear along the upper wall of the auditory canal and spreading of the pus to the squama and zygomatic process.

Anatomy.—The affected bone exhibits all the signs of carious destruction. The abscess walls are covered with granulations and the pus

is usually fetid. The bacterial examination reveals, in a large number of cases, bacteria of the coli group or of tuberculosis. The affection usually ends with complete destruction of the affected bone.

Symptoms.—Abscesses in the region of the squama and zygomatic process of the temporal bone are mostly found in chronic tuberculous suppuration of the middle ear or in tuberculous syphilitics. Examination of the ear usually reveals chronic, neglected suppuration of the middle ear, abundant granulation, and secretion of fetid pus. The development of the condition is rather insidious, painless, and without any changes attributable to the middle-ear affection. The patient notices a gradual swelling of the affected region of the skull. There may be œdema of the lid and actual displacement of the bulbus upward and exophthalmos in advanced cases. Spreading of the inflammation to the submaxillary articulation may cause transitory or permanent trismus, with a consequent difficulty in ingesting food. There are also lachrymation, conjunctivitis, and sometimes development of acute keratitis. Fever and central symptoms are usually absent, but there is diffuse headache.

The diagnosis causes no difficulties to the experienced physician, but it requires a careful examination of the ear, since neither the patient nor his relatives usually have the slightest idea that the source of the affection lies in the middle ear. The descent of the upper wall of the auditory canal, occasional atresia of the external meatus, the presence of a fistula in the lateral attic wall, the destruction of the upper wall of the osseous auditory canal, the livid discoloration of the auditory canal, and the secretion of fetid pus through the external meatus, distinctly reveal the otogenic character of the affection.

The differential diagnosis has principally to consider tuberculosis and syphilis of the cranial bones. Fracture of the skull, with regional subperiosteal hæmatoma, may likewise present a similar picture.

In multiple tuberculosis of the cranial bones other regions of the skull may likewise be tuberculous, such as the parietal bone, frontal bone, and orbital roof. Suppurative gummata in the region of the squama of the temporal bone or zygomatic process may sometimes simulate an otogenic process. Precise establishment of the history, X-ray examination, and, if necessary, examination for complement deviation, will render the diagnosis possible.

Atheromata are very rare in this region; nevertheless, an otitic abscess may exceptionally be simulated by a suppurative atheroma. The intact middle ear and the entirely unchanged auditory canal will lead to the correct diagnosis.

Regional malignant tumors need not be considered for the purposes of differential diagnosis, except sarcoma. This may be either pure sarcoma or sarcomatous mixed growths of the parotid gland (which can be

easily recognized by their source) or medullary sarcoma of the dura. All these growths are perforated in all directions by the squama of the temporal bone and seem to have fluctuation. In the latter case there are always fetid suppuration of the middle ear and tormenting headache.

Traumatic hæmatoma in the region of the squama of the temporal bone and zygomatic process after fractures occurs almost exclusively from direct trauma. Aside from the history, there are sufficient guiding points gained by an examination of the visible injuries of the neighboring skin to assure the diagnosis.

Treatment, Course, and Prognosis.—The treatment consists in the surgical exposure of the affected bone, but it should be remembered that operation in the region of the temporal squama or zygomatic process must always be preceded by antrotomy in middle-ear suppuration or by the radical operation in chronic cases. The skin incision should be made far enough forward to admit of reliable drainage of the abscess. In order to prevent disfiguring scars, the incision should always be made above the border of the hair if at all possible, and the zygomatic process be exposed by displacing the skin flaps. To expose the abscess itself great care is required. The soft layer is resected gradually and carefully, since a careless incision invites the danger of injuring the dura of the middle cranial fossa by perforating the temporal squama, which may already be impaired.

The course generally is tedious, which is not surprising, as all these patients have been weakened from inadequate nutrition. Besides, there are very often accompanying tuberculosis and syphilis.

The prognosis should, therefore, be very guarded. The course of the affection takes from two to four months; in tuberculous cases the possible subsequent development of tuberculous meningitis or cerebral tuberculosis should be taken into account.

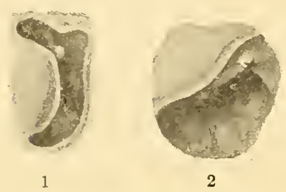
III. FISTULA FORMATION IN THE OSSEOUS AUDITORY CANAL

Fistula formation in the osseous auditory canal, as a sequel to middle-ear suppuration, occurs oftener in chronic than in acute cases. The fistula is generally situated in the lateral antrum wall. The destruction of the integument of the auditory canal, if it should take place, may not go beyond the locality of the fistula; sometimes, however, there occur extensive ulcerations of the integument of the auditory canal. The result will be the complete destruction of the membranous auditory canal and the formation of a circular ulcer. The fistula formation itself takes place in conjunction with the changes which lead to the expulsion of an antrum sequestrum or consequent upon acute ulcerative disintegration of a middle-ear cholesteatoma. The orifice of the fistula is sometimes more or less round in shape, sometimes in the shape of a fissure

running in the longitudinal direction of the auditory canal; in other cases, again, the latter shows cribriform perforation (Fig. 92).

In chronic tuberculosis of the middle ear the integument of the auditory canal may happen to remain intact in spite of a fistula if the perforation makes only very slow headway (Fig. 97). As the ulceration advances, it causes the soft parts to be gradually detached from the bone of the external auditory meatus, with consequent prolapse of the wall of the canal (Fig. 97). The canal is flooded by pus on all sides, and the pus finally perforates at the transition from the osseous to the cartilaginous auditory canal, spreading forward to the submaxillary bone. In some cases there may be a descent of pus closely below the concha at the spot covered by the lobulus. In children the forward perforation may occur through the ossification gap of the tympanic bone, leading to periarticular tuberculosis of the submaxillary articulation. This may finally cause perforation into the articulation itself.

Fig. 97.



Otoscopic findings in fistulæ of the antrum or auditory canal. 1, prolapse of the posterior wall of the canal; 2, prolapse of the posterosuperior wall of the canal.

Symptoms.—Fistulæ in the region of the osseous auditory canal may develop without hindrance. The permeability of the fistula is indicated by considerably increased secretion of pus from the external meatus and is accompanied by the destruction of the auditory canal. Small particles of bone or of the cholesteatoma are expelled along with the pus. Fistulæ with a slow course of development may be completely occluded by polypi, and in these cases manifestations of pus retention will occur in spite of the presence of the fistula. They consist in slight but very fetid secretion, headache, greatly impaired hearing acuity, and a sensation of fulness and heaviness in the ear. Should, as it sometimes happens, the secretion be entirely arrested, there may be considerable elevation of temperature.

Diagnosis.—A fistula of the auditory duct can usually be recognized in the otoscopic examination and demonstrated by the introduction of a bent button probe. Fistulæ which are located near the border of the tympanic membrane may simulate perforation of the latter. Small fistulæ which are located near the antrum wall may require careful examination for detection. The probe encounters rough bone. The manipulation of the probe requires great gentleness of action, owing to the danger of dislocating the auricular ossicles. Carelessness may also cause a paresis of the facial muscles, evidently by pressure on the exposed facial nerve. This, however, will only be transitory.

Local anæsthesia should be induced in order to save patients any inconvenience during the local examination, but general anæsthesia is not required in any of the cases.

In the presence of intracranial or labyrinth symptoms (vertigo, vomiting, equilibrial disturbances), examinations with the probe and irrigations with the tympanic tube are, of course, to be avoided. With the integument of the auditory canal destroyed and in the presence of a large permeable fistula, the correct diagnosis can be made from the otoscopic examination without resorting to instrumental examinations.

The differential diagnosis will have to consider: Bone abrasions of the external auditory canal following circumscribed necrosis of the soft covers, particularly erosions of the external canal caused by acids, such as sulphuric or carbolic acid, fracture of the external canal.

Course.—Left to itself, the fistular orifice will increase in size, the result being a complete destruction of the posterosuperior osseous wall of the canal. On examination we shall find the picture of the so-called "natural radical operation," with exposed attic and antrum. The suppuration of the middle ear, however, generally continues, and the number of cases in which the process can be arrested by conservative treatment is very restricted.

In other cases there will be formation of osteophytes in the middle ear and external canal, organic stricture of the latter, circular ulceration of the integument, and atresia of the canal which may be permanent. Should, however, the perforation of the middle ear persist, and especially in the presence of a cholesteatoma, the atresia will again be broken down for the time being, or the occlusion may persist and the periodical perforation will occur through fistula formation of the mastoid process.

Treatment.—Spontaneous healing being a very rare occurrence, operation is the indicated treatment in every case of fistula of the auditory duct. This consists in the radical exposure of the middle-ear spaces, and is a very simple procedure, considering that the osseous wall has already been partially or completely destroyed. After the plastic operation of the canal, the lividly discolored or ulcerated parts of the integument of the canal must be resected. Should any pus descend toward the mastoid process or submaxillary articulation, it will be necessary to expose the abscess region in accordance with general surgical principles.

The prognosis in non-tuberculous cases is thoroughly favorable, and identical with the prognosis of chronic middle-ear suppuration without fistula. Radiation by artificial light or sunlight often does excellent service in the traumatic treatment of tuberculous cases. As a matter of course, the physical condition of the patient should likewise be improved by adequate nutrition.

Involvement of the submaxillary articulation and simultaneous presence of submaxillary ankylosis require two operations, as in most cases the ankylosis cannot be operated upon until the radical operation has taken place.

IV. OTOGENIC DESCENDING ABSCESSES OF THE SUBMAXILLARY REGION

Pus descending toward the submaxillary articulation in the absence of a fistula of the auditory canal is exceedingly rare. It is observed in children in acute middle-ear suppuration following grave infectious diseases. In my own experience, the affected patients have not had medical attention in the acute stage, the middle-ear suppuration being left to take care of itself.

The treatment should always set in simultaneously with the surgical treatment of the middle-ear suppuration. In fresh and all such cases in which the middle-ear suppuration has not existed for more than eighteen months, antrotomy will, as a rule, suffice. The submaxillary articulation may then be exposed and the ankylosis removed at the same time. Radical operation is unavoidable where the suppuration has existed for more than eighteen months. Operative interference with the submaxillary region is not indicated immediately following a radical operation, unless there is a florid suppuration or a descending abscess.

V. DESCENDING ABSCESSES ALONG THE EUSTACHIAN TUBE

Pus descending along the Eustachian tube is the only hyperacute affection among the extracranial otitic diseases. It is rare, and appears as a complication of grave acute or chronic suppuration of the middle ear, sometimes in the course of an ichorous bulbus thrombosis. There will be phlegmonous inflammation of the soft parts of the tube, with subsequent, rapidly advancing, degenerative suppuration. The latter continues partly in the form of submucous descending abscesses toward the pharynx; partly it causes acute, purulent pharyngitis, with suppurative laryngitis or laryngeal œdema due to destruction of superficial tissue; while in cases running a particularly rapid course there will be descent of pus toward the fundus of the buccal cavity, purulent periphlebitis, and venous thrombosis.

The diagnosis of these cases is not an easy one, the less so as patients often present themselves for examination when their condition is exceedingly complicated.

The pharyngoscopic picture simulates a grave ulceration from a peritonsillar or retropharyngeal abscess, and the real cause of the trouble is only discovered by a careful examination of the auditory canal.

The surgical treatment is often aggravated by the fact that it is difficult to open the descending abscesses sufficiently from the ear trauma (antrotomy or radical operation), and a reliable drainage by counter incisions at the neck or arch of the buccal cavity can not often be established.

The prognosis is unfavorable in a large number of cases, the patient succumbing to the rapidly progressing purulent decomposition of the

soft covers of the fauces and larynx, or a pyæmia may develop with all its sequelæ. Nevertheless, there are also benign cases which recover as peritonsillar abscesses.

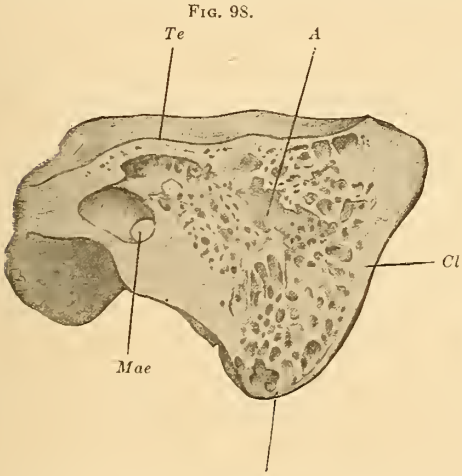
The unfavorable prognosis is based in many cases upon the presence of infections caused by highly virulent pyogenic factors.

VI. OTITIC DESCENDING ABSCESSES OF THE NECK AND SUBOCCIPITAL OTOGENIC SUPPURATION

These affections are occasioned by downward perforation of a mastoid abscess. There is an anatomical predisposition in the fact that the cortical layer is very often thicker at the lateral surface of the mastoid process than at the apex or middle wall (Figs. 98, 98*a*, 99, 101).

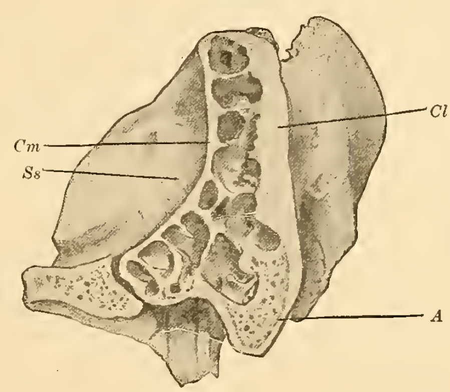
In the region of the mastoid cells especially, the cortical layer of the middle wall and apex are often found as thin as paper and transparent

Fig. 98.



Normal mastoid process. The lateral wall of the corticalis (*Cl*) is as thin as paper at the apex.

Fig. 98*a*.



Mastoid cells with diploic apex (*A*). The lateral cortex (*Cl*) is thick, the cortex of the apex (*A*) as well as the internal cortex (*Cm*) are thin. *Ss*, sulcus sigmoideus.

(Fig. 98), with the consequence that a gradually growing abscess, which reaches the cortical layer at about the same time, will perforate through the apex or middle wall (Figs. 99–101, 103) rather than through the cortical layer of the lateral wall.

Up to the age of four years the mastoid process is but slightly developed and the entire cortex is thin. In the neighborhood of the antrum especially, the osseous wall of the mastoid is but slightly resistant, which accounts for the fact that in infancy perforations of the upper part of the mastoid with formation of subperiosteal abscesses occur oftener than descending abscesses of the neck or nape by perforation of the apex (Fig. 103). Otitic descending abscesses of the neck in infancy are caused by destruction either of the fundus of the tympanic cavity or of the inferior wall of the auditory duct or by the downward spreading

of a subperiosteal abscess following a perforation of the periosteum outside the perimysium of the sternocleidomastoid muscle (Fig. 102).

Bezold was the first to study otitic descending abscesses both anatomically and clinically, and it is for this reason that mastoiditis which is accompanied by these manifestations is known as Bezold's (Fig. 99).

According to the localization of the perforation, three kinds of descending abscesses can be distinguished (Fig. 101):

Fig. 99.

Anatomy of Bezold's mastoiditis. A sequestrum is visible in the large fistular aperture (a) at the medial surface of the mastoid process.

(1) Descending abscess within the sternocleidomastoid, the perforation occurring in the region of the insertion of that muscle (Fig. 100).

(2) If perforation occurs through the internal wall of the apex, the pus will appear in the spaces between the fascial layers of the neck and nape.

(3) If the perforation occurs in the region of the insertion of the digastric muscle, the pus will invade that muscle. As the abscess grows, the digastricus will be flooded by pus, which finally descends in the direction of the tendon of that muscle, forward toward the pharynx and the arch of the buccal cavity.

Course.— Superficial abscesses located in the sternocleidomastoid (Fig. 100) grow but slowly, the degree of growth depending upon purulent disintegration of the muscular tissue of the sternocleidomastoid. The abscess is always confined to the upper part of the muscle, and causes a flattened spherical swelling in the region of the sternocleidomastoid which may be surrounded by extensive œdema (Fig. 100).

After perforation into the facial fissures, large quantities of pus will rapidly descend. The advancing pus meets with but little resistance, and may within a few days reach the roof of the pleura and the clavicle in front and the scapula and cervical vertebral column behind. Besides, widely ramified abscesses will form between the superficial and deep muscles of the neck and nape (Fig. 103), and pus will descend toward the pharynx and sometimes perforate into the pharynx itself through its lateral wall.

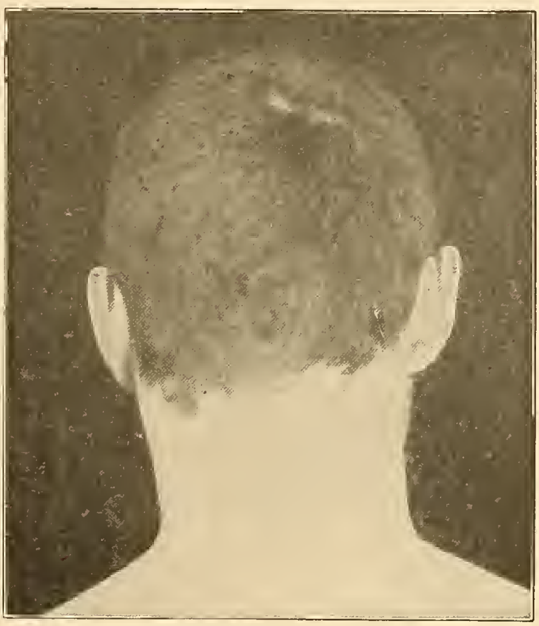
After perforation toward and into the digastric muscle the pus will descend toward the fundus of the buccal cavity, with phlegmonous inflammation of the vicinity. Here, again, a spontaneous perforation into the pharynx may sometimes occur.

Symptoms.—There is always a suspicion of a descending abscess toward the neck where in the presence of mastoiditis the apex of the mastoid is not palpable (Fig. 100). Patients with short, thick necks are an exception, as in their case the apex may not be palpable although no inflammation whatever is present.

There are all the manifestations of purulent mastoiditis. It is only in a relatively small number of cases that the inflammatory manifestations of the mastoid recede after the occurrence of a downward perforation. In rare cases the entire middle ear may completely recover, so that the mastoid, tympanic membrane, and hearing acuity are found normal.

Superficial descending abscesses are accompanied by spontaneous and pressure pain in the region of the sternocleidomastoid. The motility of the head is impaired, the head being often held in oblique position, with inflection toward the affected and torsion toward the healthy side. After a descending abscess has persisted for some time, the skin over it will be drawn tight and hyperæmic and there may also be fluctuation. In the early stage fluctuation may be entirely absent if there are considerable swelling and inflammatory infiltration of the muscle.

Fig. 100.



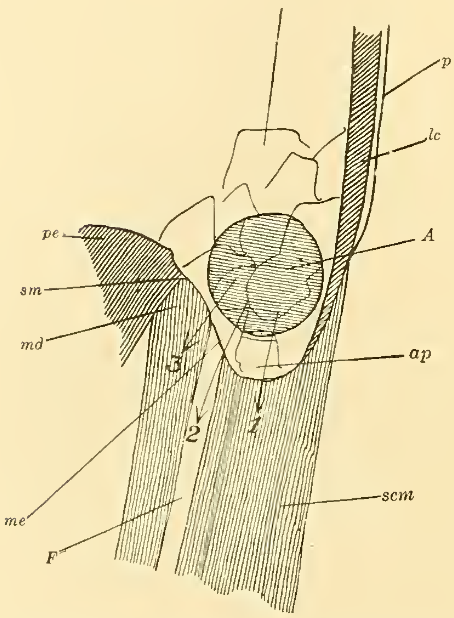
Left otitic descending abscess. The flattened spherical swelling of the submastoid region of the neck (*a*) is distinctly visible in the posterior aspect.

Pus descending along the facial fissures usually causes pain in the shoulder axillary region. This may be mistaken for muscular rheumatism if the middle-ear inflammation itself has already healed by that time. If the abscess extends anteriorly to the pharynx, it can be felt in the shape of a resistant protrusion of the lateral pharyngeal wall by digital examination from the pharynx. After perforation into the pharynx the patient feels a sweetish pus taste in the mouth and complains of anorexia. In some cases pus is expectorated or vomited. In deep perforation there is violent headache, with difficult deglutition and mastication. In the acute stage there is usually continuous fever; in later stages the temperature may be normal.

Diagnosis.—The diagnosis of superficial descending abscesses of the neck is exceedingly simple. Any previous or still existing mastoiditis, together with the fact that the pain in the mastoid was less or arrested after the growth at the neck occurred, will at once suggest an otitic descending abscess.

Where, however, perforation into the deeper layers of the neck has persisted for some time, the diagnosis may cause difficulties. Such is particularly the case if at that time the middle-ear inflammation has already abated and no abscess symptoms are demonstrable at the mastoid process. It is by no means a rare occurrence for patients to come to the ear specialist after a number of other examinations have been fruitless; patients may have been locally treated for weeks under the diagnosis of acute rheumatism or acute swelling of the lymph-glands, without as much as a thought having been given to the etiological importance of middle-ear inflammation.

Fig. 101.



Schematic frontal section through the right mastoid process, illustrating the topographico-anatomical condition in otitic descending abscesses. Natural size. *pm*, mastoid process; *lc*, lateral (thick) corticalis of the mastoid process; *p*, periosteum; *ap*, apex of the mastoid, in this case enclosed by a thin cortex; *msc*, sternocleidomastoid muscle; *F*, fascial spaces; *me*, medial corticalis of the mastoid (in this case thin); *md*, musculus digastricus; *sm*, sulcus mastoideus; *pe*, petrous bone.

The mastoid abscess (*A*) is prevented from perforating outward by the thick lateral corticalis; it therefore perforates downward either into the sternocleidomastoid (type 1), into the fascial spaces (type 2) or into the musculus digastricus (type 3), forming a descending abscess (see p. 288).

For purposes of differential diagnosis the following affections have to be considered:

(1) Acute muscular rheumatism.

(2) Acute inflammation of the deep lymph-glands of the neck and nape.

(3) Tuberculosis of the cervical vertebral column.

(4) Osteomyelitis of the cervical vertebræ.

(5) Tumors of the cervical vertebral column.

In most cases there is no difficulty. The history and examination of the neck (inspection and palpation from behind, Fig. 101) will positively reveal the otogenic character of the affection.

The condition of the cervical vertebral column is found by X-ray examination. In muscular rheumatism good motility of the head and neck for at least several hours, or possibly complete disappearance of the complaint, will be attained by hot-air treatment, while the same treatment can, of course, have no favorable effect upon descending abscesses.

The difficulties are greater in many cases of deep abscesses of the lymph-glands, especially since otitic descending abscesses may in every case cause swelling or suppurative inflammation of glands and formation of actual glandular abscesses of the neck. If the history fails to establish any affection of the ear, if tympanic membrane and mastoid

process are intact, and the hearing acuity is normal, the otitic character of the affection may be excluded, as a rule. In doubtful cases, however, the differentiation can only be made on the operative findings.

An otitic descending abscess of the neck extends from some circumscribed place to the exposed bone or upwards to a mastoid fistula, while a glandular abscess is surrounded on all sides by soft parts.

Treatment.—The treatment of otogenic descending abscesses of the neck consists in incision and evacuation. The first step of the opera-

Fig. 102.

Otitic descending abscess, the pus descending toward the nape. Four-year-old girl.

tion is always opening of the mastoid, including cases in which clinically no more mastoid manifestations can be detected. If at the time of operation the tympanic membrane is closed and there is good hearing acuity, mastoidotomy will suffice. The apex of the mastoid is then exposed, its lateral wall and base are incised, and the fistula should be demonstrated, if possible.

If the fistula is located at the apex, it can be completely removed; where it is located at the middle surface of the apex, it should be simply transformed into an open groove by removing the lateral wall, as more extensive resection of the bone invites the danger of exposing or injuring the facial nerve.

Should pus exude from the external auditory meatus at the time of operation, or the acuity be considerably reduced in spite of a closed

tympanic membrane (3½–4½ feet C.), the preliminary act of the abscess operation should consist in antrotomy, or in chronic cases, of course, in the radical operation.

In abscesses extending between the fascial laminæ, the skin incision is made very deep in order to reach the deepest point of the abscess, thus making sure that the entire abscess will be drained and further descending of pus need not be feared (Fig. 103). The descending abscess is opened by enlarging the retro-auricular skin incision.

Fig. 103.

Bezold's mastoiditis. Scars after healing of multiple, widely ramified, descending abscesses of the neck.

In abscesses within the perimysium of the sternocleidomastoid, it will be sufficient to elongate slightly the skin incision beyond the apex of the mastoid, because after evacuation of the abscess any extensive descending of pus will no longer occur, owing to the resistance of the muscular tissue. If necessary, counter-drainage may be provided at the lower end of the abscess.

If at all possible, the skin incision should be made along the anterior border of the sternocleidomastoid. Any pressure tending to expel the contents of the abscess should be avoided, all that is necessary being to provide a suitable aperture through which the pus may spontaneously flow off. The healing process will then usually run a smooth course. Should the opening be insufficient, voluminous secretions of pus will continue for days or weeks, until sudden pain and fever indicate new progress of pus descending or the formation of a new abscess.

Intramuscular abscesses, even large ones, will heal without causing motor disturbance of the head or vertebral column, except in cases where the healing process is slow and children have to wear a head and neck support. This may cause oblique position of the head, which, however, will spontaneously remedy itself or completely disappear under hot-air treatment. Massage should not be instituted for a long time afterward.

If there was perforation toward the pharynx, drainage strips should be inserted up to the aperture of the perforation. The pharyngeal fistula will heal spontaneously and completely without any local treatment in 1–4 weeks after the descending abscess has been opened.

XIII. ENDOCRANIAL OTOGENIC AFFECTIONS

The most important endocranial otogenic affections in infancy and early childhood are pachymeningitis externa, extradural abscesses, and serous, suppurative, and tuberculous meningitis. Otitic thrombophlebitis is less frequently observed in infants and children under four years of age. It appears that they acquire suppurative meningitis much more readily under the same circumstances which cause serous phlebitis in older children and adults. This may be attributable to the fact that the cranial surfaces of the temporal bone in infants and young children are much more intimately connected with the external surface of the dura than is the case in older children or adults.

The cases of cranial abscess observed in children during the first years of life are almost without exception suppurative cerebral tubercles of the third ventricle or cerebellum.

I. PACHYMENINGITIS EXTERNA AND EXTRADURAL ABSCESSES

Occurrence.—Extradural abscesses spreading between the bone and the dura always occur in conjunction with inflammation of the external surface of the dura (pachymeningitis externa); occasionally they even represent the end result, pachymeningitis externa. The cases in which pachymeningitis or extradural abscesses develop in the course of acute middle-ear suppuration are to be clinically strictly separated from those occurring in the course of chronic middle-ear suppuration. According to the length of time the dural changes have persisted, we distinguish between the acute and chronic extradural abscess. According to localization, we distinguish extradural abscesses of the middle cranial fossa and those of the posterior cranial fossa. The extradural abscesses situated between the surface of the petrous bone and the dura may be designated as paralabyrinthine; those in the area of the venous sinuses, especially the sinus sigmoideus, the middle wall of which is formed by the dura and the sinus, are called perisinous (wrongly perisinuous) abscesses.

In many cases pachymeningitis and extradural abscesses are part manifestations, the beginning or termination of some other endocranial otitic affection (sinus thrombosis, meningitis, cerebral abscess), or of a suppuration of the labyrinth. It may happen in very rare cases that a suboccipital otitic descending abscess, which has originated at the base of the brain, penetrates up to the dura through the osseous base of the skull, involving the external surface of the dura or leading to extradural abscess.

Anatomy.—Pachymeningitis externa and extradural abscesses, like other intracranial affections, may occur by direct spreading to the dura and the extradural spaces of a pus focus that may be present in the temporal bone, or by way of metastasis. In the former case the temporal bone is usually softened up to the dura, and there is already a more or less extensive osseous fistula which is permeable up to the dura.

If the changes are of metastatic origin, the temporal bone in the region of the abscess or of the extradural affection may appear macroscopically intact, in which case the lateral wall of the abscess is formed by the normal cortical layer of the cerebral or cerebellar surface of the temporal bone.

Pachymeningitis externa sets in with the secretion of a fibrinous exudate between dura and osseous surface, the first effect of which is the separation of the dura from the bone. This means that the intimate union between dura and bone which prevails in young children must be severed by the exudate. It does not require a long time for fibrinous layers to appear on the external surface of the dura. They are grayish white or yellowish white, completely cover the bluish-white color of the dura, and are coarse to the touch. The periphery of these pachymeningitis plaques is sharply demarcated and encircled by perfectly normal dura. Pus will now form with the aid of micro-organisms, accumulating between dura and bone. This accumulation, so far as its surface extension is concerned, may be restricted to the area of the original changes. In many cases, however, especially in those where the pus is under considerable pressure, the dura will be lifted up from the bone for an extensive area, and there will be extensive superficial abscesses.

In abscesses which have been caused by direct extension of suppuration of the ear there is usually from the first a communication between the extradural abscesses and the hollow spaces of the temporal bone. As in these cases the pressure in the extradural abscess is not particularly high, extensive extradural abscesses which have spread in continuity of process are very rare. On the other hand, abscesses which were caused by metastasis and are closed in from all sides may assume very large proportions, and in rare cases lead to suppuration of a large part of the dura of the posterior or middle cranial fossa on the affected side, and to secondary perforation outward.

Abscesses of the posterior cranial fossa are bounded in most cases at the level of the sinus transversus and the superior edge of the petrous bone. Exceptions, however, are by no means rare, so that a perisinous abscess of the posterior cranial fossa may spread from the knee of the sinus upward to the middle cranial fossa; or a paralabyrinthine abscess or an extradural abscess of the middle cranial fossa, situated above the

antrum, may spread beyond the upper edge of the petrous bone to the posterior cranial fossa.

The depth of extradural abscesses varies. As a rule, they do not exceed a few millimetres. On the other hand, some acute extradural abscesses of the posterior cranial fossa which descend toward the occiput may give rise to considerable accumulation of the pus at the base of the occiput. Deep extradural abscesses of the middle cranial fossa, accompanied by compression of the cerebrum (temporal and parietal lobes of the affected side), are very seldom observed. In this region there are only chronic extradural abscesses with acute exacerbation. The latter may be caused by trauma (fall, sabre cut, horse kick, etc.) or by reinfection in the course of chronic middle-ear suppuration.

If pachymeningitis externa or an extradural abscess has existed for a long time, granulations will form at the walls of the abscess. Should there be an osseous fistula, this will be first attacked by granulations, next the external surface of the dura, and finally the surface of the bone facing the dura.

Anatomical Course and Termination.—Pachymeningitis externa and extradural abscesses may heal spontaneously in the early stage of the affection. The contents of the abscess are partly resorbed and partly undergo connective-tissue organization. The granulations are gradually replaced by connective tissue. The rest of the changes consist in thickened layers of connective tissue, in deposits on the dura mater, and in a particularly intimate connection of the dura with the bone.

In the absence of resorption and spontaneous healing the extradural abscess will finally perforate in the neighborhood. Perforation outward will be favored by the presence of an osseous fistula which precedes abscess formation in cases where the abscess is the result of a continuous process. In cases of metastatic extradural abscesses the fistula may be formed at a later period.

According to locality there are to be distinguished: (1) fistula of the mastoid, (2) of the antrum, (3) of the tegmen, (4) of the labyrinth.

The mastoid fistula is located in the posterior cranial fossa at the medial surface of the mastoid, and is most frequently observed in extradural abscesses and usually causes a broad communication between the extradural and the mastoid abscess.

An extradural abscess may be evacuated into the antrum or tympanic cavity through a fistula of the antrum or tegmen.

Should an extradural abscess lead to the formation of a labyrinth fistula, it is usually one of the upper semicircular canal, and it invariably attacks a canal which is provided with a thin bony layer of slight resistance. The consequence is suppuration of the labyrinth, followed possibly by sequestration of the labyrinth.

The contents of an extradural abscess first reach the hollow spaces of the temporal bone through the fistulæ. Here the pus may either be arrested, leading to abscess formation in the labyrinth or middle-ear spaces, or it may finally perforate outward through the lateral surface of the mastoid, forming a subperiosteal mastoid abscess.

As a rule, otitic extradural abscesses which have originated in the ear perforate outward into the region of the temporal bone. Exceptions are rare. I observed a case where an otitic extradural abscess of the middle cranial fossa perforated through the sphenoid bone, the pus spreading along the inner surface of the temporal muscle and in the muscle itself. In two cases of extradural abscess of the posterior cranial fossa, perforation with formation of a suboccipital descending abscess occurred through the basal part of the occipital bone, which was very thin. In one case perforation occurred through the parietal bone.

An extradural inflammation is spread to the endocranium by fistula formation in the dura, either by continuity without a fistula or by way of metastasis. The affection terminates by infection of the venous sinuses with purulent thrombophlebitis and pyæmia, sometimes by a sinus fistula, by pachymeningitis interna, pachyleptomeningitis, encephalitis, or cerebral abscess.

Symptoms. Clinical Course.—Pachymeningitis externa and extradural abscesses of the middle cranial fossa are in most cases associated with localized headache. The pain is of a stinging nature and restricted to certain regions of the head, which, however, do not coincide with the anatomical seat of the lesion. The pain may even be referred to the opposite side of the skull.

Affections of the external surface of the dura mater of the posterior cranial fossa are likewise accompanied by pain. This is usually referred to the mastoid region and is then a part manifestation of the other mastoid symptoms. There is also pressure pain immediately behind the mastoid, which is an exceedingly characteristic sign for pachymeningitis externa and extradural abscesses of the posterior cranial fossa. Sometimes there is also œdema in the same region or pain in the nape of the neck.

There is moderate fever in acute abscesses, while in older and extensive abscesses or in extradural abscesses which have perforated outward the temperature may be perfectly normal.

Extradural abscesses which are accompanied by considerable compression of the brain may lead to manifestations of cerebral pressure and infected foci, in exceptional cases even to all the symptoms of a suppurative pachyleptomeningitis and cerebral abscess. In otitic extradural abscess of the middle cranial fossa, for instance, spasms of the facial muscles and extremities are not infrequently observed. Large extradural abscesses on the left side may cause temporary disturbances of speech

in right-handed people (amnestic aphasia). Large acute extradural abscesses of the middle cranial fossa, which have grown rapidly, may cause unconsciousness and delirium; large extradural abscesses of the posterior cranial fossa may cause disturbance of coördination or abducent paralysis on the affected side. The fundus of the eye is normal in pachymeningitis externa, small abscesses, and where an extradural abscess communicates with the middle-ear spaces. Closed extensive extradural abscesses, however, may lead to bilateral venous stasis of the fundus, and examination will show pathological repletion of the veins. Pronounced choked disk, however, is not of frequent occurrence. The fluid obtained by lumbar puncture is normal in many cases, but in extensive subacute or chronic extradural abscesses it is sometimes turbid. It will show very minute fibrin coagulation in from six to twenty-four hours, and the microscope reveals varying quantities of mononuclear and polynuclear leucocytes, but is sterile both under the microscope and in culture.

In extensive abscesses of the posterior cranial fossa which extend to the neighborhood of the foramen magnum or have perforated outward extracranially there will be oblique position of the head toward the healthy side, impairment of active and passive motility of the head and cervical vertebral column, and sometimes stiffness of the neck.

Diagnosis.—The pathological manifestations of extradural abscesses are divided into three stages,—(1) the initial, (2) the latent, (3) the manifest stage.

The first stage is characterized by headache, which in abscess of the posterior cranial fossa is experienced as pressure pain immediately behind the mastoid. The cerebral symptoms above referred to occur in large extradural abscesses. In many cases there is moderate elevation of temperature.

After two or three weeks the latent stage sets in. The temperature has usually returned to normal, and, unless there is considerable pressure in the abscess, subjective complaints may be entirely absent. Extradural abscesses at this stage are usually discovered accidentally on the occasion of some other ear operation.

The acute stage occurs quite suddenly, the manifestations setting in without warning and causing acute reinfection of a chronic middle-ear suppuration. Any kind of trauma may be the means of changing the latent stage into the acute under grave symptoms.

The experienced physician will have no difficulty in diagnosing the acute stage, as there are characteristic signs revealing an affection of the external surface of the meninges,—above all, localized headache, pressure pain immediately behind the mastoid, and perhaps œdema. Examination of the auditory canal will likewise furnish important guides, as nearly all extradural suppurative inflammations in the region of the

middle cranial fossa are associated with suppuration of the antrum and attic. On the other hand, acute extradural abscesses and pachymeningitis of the posterior fossa are nearly always accompanied by inflammatory changes of the mastoid, although there need not be pronounced mastoiditis, nor even an abscess.

In other cases the very absence of certain symptoms will suggest the correct diagnosis. Thus, symptoms with cerebral foci, which are not accompanied by any great elevation of temperature or delirium, will suggest extradural abscess; the same holds good for isolated abducent paralysis or other manifestations of a cerebellar abscess without any symptoms of acute suppuration of the labyrinth.

An exceedingly valuable diagnostic symptom of extradural abscess and pachymeningitis is furnished by the operation, provided the bone at the dura is softened or fistulous. Having opened the mastoid, or the antrum in abscesses of the middle cranial fossa, pulsating pus will be found. This is the cerebral pulsation which has been communicated to the pus by the exposed dura. In the presence of this symptom, it is always necessary to remove the softened bones and freely to expose the affected dura.

As to the *differential diagnosis* of pachymeningitis externa and extradural abscess, all other intracranial otitic affections have to be considered.

The differential diagnosis between extradural abscess and thrombophlebitis of the sigmoid sinus presents no difficulties in most cases. If no chills have preceded and there is no intermittent fever, the clinical symptoms which point to an otitic involvement of the posterior cranial fossa may with certainty be referred to extradural abscess, to the exclusion of a sinus involvement.

On the other hand, it is impossible to differentiate between an extradural abscess at the level of the dura and one at the level of the venous sinus, because a perisinous abscess does not differ in treatment or prognosis from other extradural abscesses. The inflammatory changes of the external surface of the venous sinus are never accompanied by pyæmic manifestations. An extradural abscess may represent the termination of an inflammatory sinus thrombosis, provided the thrombotic contents of the sinus are suppurative and the endosinus abscess has perforated outward between dura and bone. There will be no difficulty if a precise history is available from the beginning of the affection. Should, however, these data be insufficient or entirely wanting, no other diagnosis can be made but that of extradural abscess, while the underlying cause, the question of any preceding thrombophlebitis and the presence of an endosinous abscess which has perforated outward, can only be established at the operation.

It is possible for cerebellar symptoms to occur in the course of large extradural abscesses, but, from the fact that the former are nearly

always accompanied by suppuration of the labyrinth, the absence of such suppuration will point the way to the diagnosis of extradural abscess.

Pachyleptomeningitis can be differentiated by the temperature. Moderately elevated or normal temperature points to extradural abscess, high fever to pachyleptomeningitis. Furthermore, the exudate obtained in lumbar puncture in suppurative pachyleptomeningitis contains microorganisms; that in extradural abscess may be clear or turbid, but is always sterile.

The differential diagnosis between extradural abscess of the middle cranial fossa and abscess of the temporal lobe may give rise to difficulties, as many advanced cases of cerebral abscess are complicated by involvement of the external surface of the dura and actual extradural abscesses. Long persistence of the symptoms (for weeks or months) points to extradural against cerebral abscess. In the latent stage of a temporal abscess an exact differentiation can only be made at the operation. In abscess of the temporal lobe there will be circumscribed, more or less grave disturbance of the circulation (stasis, thrombosis), while in extradural abscess the blood-vessels of the dura are unchanged.

The clinical differentiation between extradural abscess and tumor of the dura is easy in the absence of any suppurative involvement of the middle ear, so that there is no cause for an abscess to form. In these cases the first thought should be of a tumor. Sometimes, however, an extradural tumor develops on the soil of an old middle-ear suppuration, and in these cases which involve sarcoma of the dura a clinical differentiation before operation is practically impossible. The time of operation is certainly early enough to make a diagnosis of tumor, as the temporal bone is not found to be softened from suppuration.

It is only in very rare cases that symptoms of extradural abscess can be caused by cholesteatoma of the dura. The clinical observations of such a case were recently reported by Frey.

It is important to bear in mind that the value of the clinical differential diagnosis should not be overestimated. As in all other diseases, it is not so much a question in otitic intracranial affections to diagnosticate their anatomical character and depth toward the brain, but to recognize the affection before operation. It is a perfectly satisfactory achievement to make a diagnosis in doubtful cases of a pathological involvement in the region of the middle or posterior cranial fossa. This diagnosis is a sufficient indication for immediate operation, because an imperfect clinical diagnosis may be supplemented by the operative findings.

Treatment.—The fact of a middle-ear suppuration having led to a pathological involvement of the external surface of the dura or to an extradural abscess is a sufficient indication for immediate operative

interference. Spontaneous healing of extradural abscesses and pachymeningitis is too rare an occurrence to be therapeutically foreseen with the slightest justification. Conservative treatment and a waiting attitude are still less justifiable.

Operation.—Surgical exposure of the affected dura is done through the ear. The mastoid is opened, and, if the underlying middle-ear suppuration is acute, antrotomy will be sufficient. In chronic suppuration of the middle ear, however, resection of the dura must be preceded by the radical operation. Operations in the region of the dura can only be done after the ear operation has been completed. In extradural abscesses caused by suppuration of the labyrinth, the affected dura must be exposed by resection of the affected part of the petrous bone. In operations on the dura itself as well as on the venous sinus, care should be taken that the bone is removed flat before the actual exposure of the dura or sinus is undertaken. The reason is this: After the dura has been opened with the chisel at a circumscribed place, the further exposure can be accomplished with the bone-forceps from the aperture made or through an existing fistula, so that the dura will become visible, if possible, on the level of the entire surface of the bone and not through a deep, funnel-shaped opening made with the chisel (Fig. 111). The direction of the chisel should be as near a tangent to the dura as possible.

If the bone is removed with the bone-forceps, it is necessary first to remove carefully all adhesions between the bone and the dura by an elevator or a button probe, in order to prevent an accidental injury to the dura with the chisel or the bone-forceps. The exposure of the dura, if necessary beyond the region of the temporal bone, should be continued far enough to render the entire affected region of the dura freely visible in such a manner that it is surrounded by at least 5 mm. of normal dura. It is only by observing this rule that an incomplete exposure or evacuation can be prevented. It will also guard against the danger of the abscess spreading, in spite of the operation, along the surface or even penetrating intracranially.

By correctly planning the operation the surgeon is to a certain extent independent from the clinical diagnosis. It has already been mentioned that, aside from the extradural abscess, there may be another endocranial affection which is clinically covered by the symptoms of an extradural abscess and may therefore escape attention. This applies especially to an extradural abscess which occurs in the course of a chronic middle-ear suppuration. Such a complication, however, is sure to be discovered by a systematic arrangement of the operation, and in this way I have repeatedly been in a position to demonstrate at the operation the clinically diagnosticated extradural abscess and incidentally discover an intrameningeal or cerebral abscess. If the abscess extends over

a large area, the surgeon should not hesitate to carry out an extensive removal of the bone,—to the parietal bone, the temporal squama, the occipital bone, and, in rare cases, to the large sphenoid wing.

The cavity is drained with iodoform wicks, but the principal wound is left open. If the healing process takes a favorable course without temperature, the first change of bandage is made on the sixth day after operation, the wicks being simultaneously shortened. This is repeated with each change of bandage, so that the drains introduced at the operation will be completely removed at the third or fourth change of bandage. The first change is made with the patient in the recumbent position, the operating room being most suitable for the purpose. The retro-auricular skin incision may be closed on the tenth day by sutures, with the exception of the middle third.

Should pain, fever, or chills occur after the operation, the bandage should be immediately changed.

The prognosis of pachymeningitis and extradural abscess is quite favorable, provided the operation has been done in time and correctly. Healing occurs by connective-tissue scars. The bone defect caused by the operation remains unchanged for the time being. In young and otherwise perfectly healthy individuals there will in course of time be new-formation of bone from the external cortical layer of the mastoid, which will partly fill up the gap. There will be but slight new-formation of bone of the internal cortical layer of the temporal bone.

After the healing process has taken its course, the periosteal scar will be sufficiently firm and resistant to render the use of prostheses or pelotes superfluous.

II. OTITIC THROMBOPHLEBITIS, OTOGENIC PYÆMIA, BACTERÆMIA, AND TOXÆMIA (SEPTICÆMIA)

(Synonyms: Otitic sinus phlebitis and sinus thrombosis.)

Otitic thrombophlebitis is clinically a very important affection among the intracranial diseases occurring in the course of suppuration of the ear. It occurs much more frequently in acute than in chronic middle-ear suppuration.

Etiology.—Thrombophlebitis is caused either by the suppurative middle-ear inflammation spreading to the venous sinuses or by way of metastasis; in the former case the anatomical signs of purulent inflammation can be demonstrated by continuity of process.

It is a questionable point whether toxins secreted by the inflamed region into the neighborhood can lead to suppurative inflammation of the cerebral sinuses.

Introductory Anatomical Remarks.—The venous sinuses are contained in the dura mater and form a network which separates the ear

from the cerebral cavity. This net is composed of the centrally situated sinus cavernosus; the sinus sigmoideus and sinus transversus, which fuse with the periphery; the sinus petrosus inferior, sinus petrosus superior, and the sinus petrosquamosus (present in children, but usually absent in adults), which run over the petrous bone.

The part where the sinus sigmoideus fuses with the vena jugularis likewise shows close topical relations to the ear. The bulbus venæ jugularis, which is accommodated in the fossa jugularis, lies under the fundus of the tympanic cavity. The venæ condyloideæ inosculate into the bulbus direct. Immediately below the bulbus medium-sized veins empty into the vena jugularis and form a direct communication between the veins of the vertebral canal and the jugularis interna. Neither the cerebral sinuses nor the vena jugularis interna possess any valves.

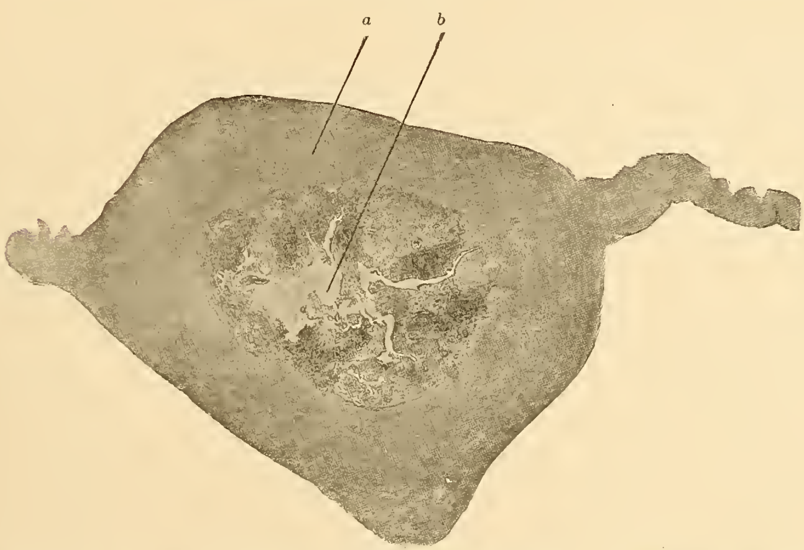
The vena facialis communis is the only one of those inosculating somewhat more centrally into the vena jugularis interna which is of any interest for our purposes. It communicates with the jugularis at the transition between the middle and upper third of the neck, but there are considerable deviations in altitude as to the place where this communication takes place.

The vena jugularis itself crosses in its course the sternocleidomastoid; in the middle third of the neck it lies immediately behind the anterior border of the muscle, in the lower third near the posterior border of the muscle, and in the upper third in front of the sternocleidomastoid muscle. Interposition of glands and fascia, together with the nervus accessorius, effect a separation at the latter place, which consequently lies in the deep layer of the soft parts of the neck. The vena jugularis is more superficially situated in the middle third and is separated there from the medial surface of the muscle merely by the fascia of the vasculo-nervous tuft. In the latter will be found the vena jugularis and the carotid at about equal depth, the pneumogastric nerve slightly deeper, and the sympathicus considerably deeper. A cross section of this bundle shows that the jugularis is completely isolated in it by a thick vascular sheath from the carotid and the two nerves, so that the vein can be completely brought to view without exposing the two nerves and the carotid.

Introductory Physiological Remarks.—The blood-pressure in the vena jugularis and the regional cerebral venous sinuses is not great under normal conditions. It is even negative when the head is held erect and a position of deep inspiration assumed. It is higher and positive in the recumbent position and drooping head. The blood stream in the cerebral venous sinuses is directed toward the foramen jugulare under normal conditions, so that for instance the sinus blood of the right side of the head escapes through the right bulbus and the right jugularis

interna. The current is stronger in the axis of the vessel than in the periphery; in the immediate proximity of the sinus wall the blood has but little motion, although the possibility of currents at the knee of the sinus sigmoideus and the head of the bulbus jugularis is admitted. The vena jugularis collapses more or less completely under negative pressure, but the venous sinuses and bulbus remain permeable through normal fixation of their lateral wall. An empty venous sinus always indicates a peripheral occlusion preventing the supply of blood. The sinuses can only collapse under negative pressure or after evacuation of the blood

Fig. 104.



Cross-section through the sinus sigmoideus in infectious thrombophlebitis. *a*, considerable callous thickening of the sinus wall; *b*, remnant of lumen.

they contain, if the lateral sinus wall has been detached from the bone and becomes movable through pathological processes (pachymeningitis, extradural abscess).

Pathological Anatomy.—The following inflammatory changes have to be distinguished: (a) the sinus wall, (b) the sinus contents, (c) the immediate surroundings of the sinuses.

(a) The changes of the sinus wall correspond to the various stages of the phlebitis. As soon as the inflammation has spread by way of continuity from the region of the éar to the sinus, they set in with inflammatory infiltration and suppuration of the external connective-tissue layers (suppurative periphlebitis). In some cases a perisinous (extradural) abscess develops between the lateral sinus wall and the bone. Periphlebitis leads to inflammation of the venous wall itself and finally to destruction of the endothelium (Fig. 104). The destruction of the endothelium is followed by partial (parietal) or complete (obturat-

ing) thrombosis of the sinus contents. The inflammation may also spread to the medial sinus wall and to the medial surface of the regional dura mater (pachymeningitis interna). Should the thrombotic contents be suppurative, they will perforate outward and cause a fistula to form at the lateral wall. A rare contingency consists in fistula formation at the medial surface of the sinus, causing either an intrameningeal abscess or diffuse suppurative meningitis.

(b) The changes of the sinus contents are characterized by the inflammatory thrombosis, which is caused in the first place by the destruction of the endothelium at the inflamed place of the sinus wall.

Fig. 105.

Obturating, infectious, fusiform thrombus with pointed ends. Natural size. Removed by operation from the sinus sigmoideus. Otogenic pyæmia with infectious thrombosis of the jugularis (see Fig. 110) in the course of a middle-ear suppuration complicated by ichorization of a cholesteatoma. Recovery.

Fig. 106.

Infectious thrombus, natural size, removed by operation from the sinus sigmoideus of a nine-year-old boy. Otogenic streptococcus pyæmia with pachymeningitis in the course of acute scarlatinous otitis. Recovery.

Fig. 107.

Thrombus, natural size, removed by operation from the sinus sigmoideus, sinus transversus, and the bulbus jugularis. Pyæmia in the course of a subacute suppuration of the middle ear. Recovery.

The thrombus is rather adherent to the inflamed parts of the wall. It may be flat and grooved, so that the sinus remains permeable for the blood-current (parietal thrombosis), or the entire cross section of the sinus may be filled up by the thrombus (obturating or occluding thrombosis, Figs. 105–109).

The thrombi of the sinus sigmoideus and sinus transversus are usually fusiform in such a way that the thrombus is attenuated at both ends (Fig. 105). An obturating thrombus, therefore, can only completely occlude the medial section of the sinus. The thrombi of the bulbus and vena jugularis are effusions of the affected parts of these vessels (Figs. 107–109). Otitic thrombosis of the sinus cavernosus is exceedingly rare.

The color of fresh thrombi is dark red, of older ones grayish red, and of suppurative ones yellowish green. Thrombi are but seldom sterile. As a rule, they are infectious from the very beginning, micro-

organisms being found along their entire length, but sometimes only in their obturated parts or at both ends.

The color does not furnish any indication of the degree of infectiousness. Fresh, deep-red thrombi may contain pure cultures of streptococcus pyogenes, while in old yellow or yellowish-green thrombi the micro-organism may already have been destroyed and the thrombi prove sterile both in cultures and animal experiments.

The inflamed internal surface of the sinus is deep red in the early stage, brownish black or yellowish green in later stages. The external wall is thickened by fibrinous deposits, has a grayish-red or yellow color, and feels hard and coarsely elastic to the touch. In advanced stages of the inflammation it becomes discolored and friable.

Fig. 108.

Thrombus, natural size, removed by operation from the sinus transversus in extensive infectious thrombophlebitis in the course of chronic suppuration of the middle ear. Recovery.

(c) Changes of the Surrounding Parts.—Very frequent concomitant manifestations of thrombophlebitis are pachymeningitis externa on the level of the affected venous sinus and on the external surface of the dura mater in the immediate proximity, callous thickening of the sinus wall, and accumulations of pus between the dura and the bone. The occurrence of pachymeningitis interna has already been referred to.

Fig. 109.

Infectious thrombus, natural size, removed by operation from the vena jugularis interna in chronic suppuration of the middle ear. Recovery. *a*, continuation of the thrombosis into the places of inosculation of the vena facialis communis, and *b*, into one of the thyroid veins.

In some cases the cerebrospinal fluid undergoes changes. It becomes turbid, has sometimes a grayish discoloration, and shows minute fibrinous coagulations in the test-glass after standing undisturbed for 6–24 hours. Microscopic examination reveals abundant mononuclear and polynuclear leucocytes, but no micro-organisms, the fluid proving sterile both in cultures and animal experiments. There is no doubt that these cases, which represent about 10 per cent. of all forms of thrombophlebitis, are early stages of meningitis. A further proof of this being so is the presence of circumscribed acute cerebral œdema and hyperæmia of the pia mater in all cases.

The inflammatory changes of the bone itself extend in a number of cases from the middle-ear spaces to the region of the affected sinus, while in metastatic thrombosis the wall of the bone may be macroscopically unchanged. In all cases the affected parts of the sinus wall and the external surface of the dura are sharply demarcated against the normal surroundings.

The most frequent seats of insulated thrombophlebitis are the sinus sigmoideus, bulbus jugularis, and sinus petrosquamosus, in the order named. With a few rare exeptions, infection of the other venous sinuses of the auricular region can only occur by the inflammation spreading from the three sinuses named. Thrombosis may spread to all venous sinuses of the skull when the affection persists for a long time. The consequence may be that all the venous sinuses of the auricular region may become occluded, while the other sinuses contain fibrinous coagulations and organized connective-tissue layers, but still retain a central lumen as thin as a thread. Furthermore, thrombosis may spread from the sinus sigmoideus or bulbus jugularis downward to the vena jugularis interna, the ventricle of the heart, and, in some cases, to all the small veins which communicate with the jugularis.

Fig. 110.

Vena jugularis interna, filled with thrombus masses and pus, extirpated together with the bulbus. Natural size. Recovery. The wall of the vein is considerably thickened by periphlebitis and endophlebitis. The specimen is opened at two places, *a* and *b*, exposing the thrombus masses which occlude the vein. Same case as Fig. 105.

The primary affection in these cases is thrombosis itself, while inflammation of the venous wall occurs only secondarily or not at all. In fully developed cases the thrombosed jugular feels like a thick resistant cord, which is distinctly palpable from without and is connected with its surroundings by fibrinous callosities (Fig. 110). This is usually complicated by infiltration of all deep glandular bundles of the neck, and the final result may be perforation of the vein and formation of extensive fetid abscesses.

As has been shown by animal experiments, parietal sterile thrombi may be resorbed and the endothelium of the vein regenerated. In advanced cases healing occurs through organized connective tissue in the thrombus, or the latter disintegrates from suppuration with consequent formation of an endosinous abscess. In cases where such an abscess is insulated against the vascular lumen by sterile obturating thrombus ends or by connective tissue, it usually perforates outward, forming a perisinous abscess. Suppurative meningitis develops less frequently. Should, however, the suppurative thrombus be insufficiently insulated against the vascular lumen, or not at all, the result will be that infectious parts of the thrombus, which have been liberated by the suppuration, will find their way into the circulation; pus and micro-organisms will follow suit and lead to pyæmia, bacteræmia, or toxæmia.

Symptomatology.—Otitic thrombophlebitis presents highly characteristic manifestations which, barring a few rare exceptions, render the diagnosis possible at every stage of the disease.

The secretion in acute cases usually continues for a long time, but irregularly, it being alternately abundant, slight, or absent. In chronic cases the pus is fetid.

There are earache and headache, especially in the occipital and mastoid regions, but in some cases these complaints are entirely absent. The mind is clear. There are manifestations of the central nervous system, except in old cases which are already complicated by meningitis. In the latter cases there is increased cerebral pressure (headache, nausea, vomiting, restlessness, numbness, delirium, slow pulse, paralytic manifestations, plethoric veins of the fundus oculi). Active and passive motility of the head and neck are only diminished in cases where the phlebothrombosis has already spread to the bulbus or the internal jugular. There is often subicteric discoloration of the skin and scleræ. Fully developed icterus, however, occurs but very rarely.

The temperature curve is of the pronounced intermittent febrile type and is a characteristic pathological symptom. After the temperature has been normal for several hours, chills will occur, followed by a rapid rise of the temperature up to 104° or more. These attacks may occur only once or several times during the day, or they may be absent for several days together. The number of chills vary. There are cases in which there was but one attack at the onset of the sinus affection, while in others they may reach a total of twenty or more.

In old and advanced cases of thrombophlebitis most patients give the impression of being very ill, while in the early stage or in chronic cases running an insidious course any particular general manifestations may be completely absent. These latter patients will apply to the clinic for outdoor treatment, as they and their friends fail to understand the importance of the chills, and febrile accessions of temperature may entirely escape them. It should be specially emphasized that patients suffering from such a grave affection need not necessarily present grave or any general symptoms at all.

A simple thrombosis of the jugular may escape detection on palpation, as it can only be recognized by the plethora of superficial veins through a tender skin. The vessel can only be palpated at the anterior part of the neck as a thick cord in cases of purulent inflammatory thrombosis of the jugular with disintegration of the thrombus and with periphlebitis.

Exophthalmos will occur in the case of very extensive thrombosis of the cerebral venous sinuses.

Diagnosis.—The diagnosis of thrombophlebitis may be made at every stage of the disease, provided a reliable history of the disease and its course is available.

A correct valuation of the symptoms may render an early diagnosis possible, usually at the first examination.

As in the other intracranial otitic affections, care should be taken not to obscure or destroy the clinical pathological picture described by the patient, which is usually done in a distinct and well delineated manner. In the absence of serious ear symptoms there is danger of erroneously referring chills, intermittent fever, and icterus to other diseases, such as influenza, central pneumonia, gastric and intestinal affections, etc., without considering the possibility of these symptoms emanating from the ear, or assuming that any sinus affections have already run their course. This would lead to the mistake of adopting a very dangerous waiting attitude, and, by the time that the medical examiners have agreed upon the fact that the pathological picture corresponds to an otitic complication, the favorable moment for a successful operation on the ear may have passed.

On the other hand, the diagnosis may present great difficulties in cases with an incomplete, unreliable, or negative history, especially in regard to the variations of temperature. But a single sign may even in these cases suggest the presence of thrombophlebitis, such as restlessness, ill appearance, subicteric discoloration, etc.

A careful specialist will be guided by his experience, and find his suspicions confirmed in the great majority of cases when they come to operation. In those rare cases which run an afebrile course, the diagnosis can only be made through a mastoid operation which is usually performed from the indications of an acute suppurative mastoiditis.

For purposes of *differential diagnosis* in childhood, the following affections come in for consideration: (1) Uncomplicated cases of acute otitis media taking an atypical course; otitic descending abscesses with manifestations of retention. (2) Angina. (3) Pneumonia. (4) Malaria. (5) Typhoid. (6) Certain initial stages of acute infectious diseases. (7) Otitic meningitis or cerebral abscess.

(1) These are always cases of beginning otitis media after the perforation has occurred quite recently or a short time ago. Acute otitis media in childhood may set in with chills and intermittent fever, but the correct diagnosis can be made after a few hours, or, at the most, in one or two days. The fever either assumes a continuous type in a short time or entirely disappears; the chills are not repeated; abundant secretion is evacuated through the perforation. Should, however, in the further course of acute suppuration of the middle ear or in chronic cases, temperatures of 101½° or more be reached, with the picture of intermittent fever, the clinical possibility of thrombophlebitis should be taken into consideration.

In otitic descending abscesses with pus retention, high fever may suddenly set in which may have a slightly intermittent character. The

correct diagnosis follows from the absence of all other pyæmic symptoms (chills, icteric or subicteric discoloration) and from the local signs of the descending abscess.

(2) In cases of acute otitis media which are complicated by a cervical inflammation presenting pyæmic symptoms, the differential diagnosis may be embarrassing. The symptoms must be referred to the ear, if irregular or absent secretion coincides with the period of increased temperature or chills. In all these acute cases there are always simultaneous manifestations of acute mastoiditis.

(3) Pyæmic symptoms in pneumonia are only present if the affection is of a severe type and has been very extensive from the first. In examining the thorax and lungs, it will therefore be necessary to demonstrate correspondingly severe lesions. We cannot be content with uncertain pulmonary findings or with the assumption of a central pneumonia which cannot be demonstrated by auscultation. Pyæmic symptoms in such cases are rightly referred to the ear or the regional venous sinuses.

(4) The differential diagnosis as to malaria is established by microscopic examination of the blood. In all cases of septic thrombophlebitis the number of leucocytes are considerably increased, which is not the case in malaria.

(5 and 6) The differential diagnosis as against acute infectious diseases, especially in children, demands adequate experience in the diagnosis of these diseases. In the absence of such experience, a competent pediatrist should be called in for consultation. The early diagnosis of these infections is by no means so difficult as to prevent an immediate differentiation from otitic endocranial affections.

(7) The differentiation of thrombophlebitis from simultaneously existing intracranial otitic diseases does not offer any difficulty in simple cases, since the former is characterized by the absence of any cerebral symptoms. But it should be remembered that meningitis or perforation of a cerebral abscess may set in with chills. The urgency of these cases does not permit of observing whether the fever is intermittent, as is the case in meningitis and cerebral abscesses, or whether it is continuous. Besides, thrombophlebitis, meningitis, and cerebral abscesses may coexist. From a practical point of view, however, the differential diagnosis in such a case is only of subordinate value. The only requirement is to diagnosticate the fact of there being an intracranial disease of an otogenic character, this being a sufficient indication for immediate operation. The exact differential diagnosis can then be made at the operation, as there will be no difficulty in recognizing even a combination of several intracranial affections. Otological surgery here shares the same stand-point as general surgery. It is often necessary to be content with having localized a surgical disease sufficiently to permit of

instituting surgical measures at the right place, and the operative findings will furnish the exact diagnosis.

Treatment of Otitic Thrombophlebitis.—Spontaneous healing of this affection is an exceptional occurrence. It may take place by resorption of the pus and gradual obliteration of the affected parts of the sinus by connective-tissue formation if the affected thrombus in the sinus is protected at both sides by sterile thrombus masses. But even in these cases the more frequent course is for the infected contents of the sinus to perforate outward and for a perisinous extradural abscess to develop. But again there is some possibility of spontaneous resorption of such an abscess or its perforation into the mastoid, notably if the medial mastoid wall had been softened by suppuration or perforated by a fistula previous to the occurrence of sinus-phlebitis.

Spontaneous healing of thrombophlebitis is so rare that it does not warrant rational conservative treatment. It is a surgical disease which offers no chances for recovery except by timely and adequate operation. If left to itself, patients usually succumb to the sequelæ of the sinus-phlebitis, otogenic pyæmia, bacteræmia, or toxæmia, suppurative meningitis or cerebral abscess, or parenchymatous degeneration, notably of the large glands (liver, spleen, kidneys) and the heart muscle. As the danger of otogenic pyæmia is present in all cases of sinus-phlebitis, and as it is impossible to say in any particular case whether otitic pyæmia has already set in, it is necessary to carry out the operation to the same extent as in the case of developed otitic pyæmia. The object of the operation in the latter case is, of course, to eradicate the pyæmia, thereby preventing the further spreading of pus and the formation of metastatic abscesses. In thrombophlebitis without demonstrable metastases, without manifest pyæmia, however, the far more important object is to plan the operation in such a way as to prevent the formation of pus foci and metastases. Thus, it is often difficult to separate clinically otitic thrombophlebitis from otitic pyæmia, but surgically such a separation is entirely out of the question.

Plan of Operation

The demands to be made upon an ear operation are the following:

(1) Cleansing the pus focus as completely as possible and evacuation of the pus.

(2) Reliable drainage of the pathological focus.

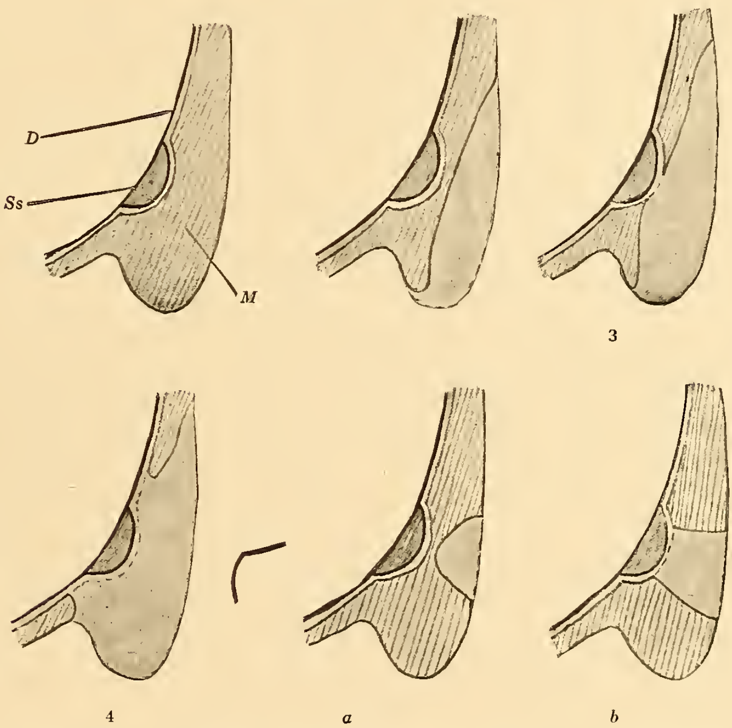
(3) Exposure and opening of the affected venous sinus and removal of the thrombus.

(4) Preventing the pus from entering the blood current. Where metastases are already present, the further spreading of the pus and formation of new metastases should be prevented as far as lies in our power.

The first demand is met by antrotomy in acute affections and by radical exposure of the middle-ear spaces in chronic cases. The surgical trauma is enlarged by two skin incisions in a posterior direction, permitting of an untrammelled survey of the affected regions of the ear and sinus.

Previous to exposing the sinus and dura, it is advisable to remove the bone of the entire surroundings close to the level of the dura, so that the chisel in the opening process may not strike the sinus and dura in a right or blunt angle, but in the direction of a tangent (Fig. 111). This

Fig. 111.



Schematic representation of exposure of sinus and dura. *D*, dura mater; *M*, mastoid process; *Ss*, sinus sigmoideus.

The correct technic is shown in Figs. 2, 3, 4. The bone is chiselled flat (2), so that the chisel may expose the sinus in the direction of a tangent (3) and the aperture thus made can be conveniently enlarged with the bone-forceps after lifting of the dura (4). The wrong technic is shown in Figs. *a* and *b*: Formation of a funnel-shaped opening (*a*), with the depth of the funnel directed toward the sinus. To reach the sinus wall involves considerable danger of injuring the sinus, as the chisel advances vertically toward the sinus. But, if the sinus wall has been reached, it will be found at the bottom of the funnel in such a small area that it cannot be conveniently surveyed (*b*).

will avoid injury to the sinus and a highly unpleasant hemorrhage of the sinus in parietal thrombosis; at the same time it will facilitate the survey of the operative field and the technic of the subsequent exposure, as it is possible to remove the surrounding bone with the bone-forceps from the aperture made. When exposing the affected part of the sinus,

it is also necessary to allow of a survey of the healthy surroundings beyond the borders of the affected area. The presence of normal bone should not prevent the surgeon from exposing the sinus and exploring the seat of the disease, as long as there are signs of sinus-phlebitis. Previous to removal of the bone, the connection between dura and bone and that between sinus wall and bone are severed with a blunt probe. This is effected by advancing the button probe from the exposed parts of the dura and sinus along the entire opening a few millimetres underneath the bone. Mobilization of the dura and sinus will prevent accidental injuries with the bone-forceps. For the same reason it is advisable to use a bone-forceps with rounded points, the best being a bent Luer's forceps. This applies particularly to the exposure of the sinus itself. The exposure should not be limited to the sinus alone, but should also include the dura both before and behind the sinus. I am in the habit of proceeding in a thoroughly systematic way, starting from the exposed region of the sinus and proceeding first to the part of the dura behind and then to that in front of the dura. After that, the endocranial course of the sinus can be exposed.

In following the sinus downward toward the bulbus venæ jugularis, great care is required, as the lateral sinus wall is comparatively thin at that place and intimately adherent to the bone, even in the adult. The adhesions must, therefore, be very carefully loosened. It is also advisable to protect the lateral wall of the sinus during the bone work by flat, fitted gauze strips, so as to prevent accidental injuries. Exposure in all the four directions named should continue beyond the affected area, until a rim of 5–10 mm. in width of normal sinus and normal dura is present in the field of operation, aside from the affected parts. The reason why the dura should be exposed both in front and behind the sinus is that most cases of thrombophlebitis, notably the advanced ones, are complicated by perimeningitis externa or even by extradural abscesses. Besides, it is technically simpler to construct a roundish aperture than one in the shape of a canal or groove that is adapted to the direction and width of the venous sinus. Technically, the bone-forceps is preferable to the chisel in the entire work. I have never seen any advantage accruing from the use of the saw, as the resulting pap-like mass interferes with the exact control of the operative field, it being often impossible, while at work, to determine whether the affected bone is completely removed. Besides, even employment of the greatest care in operating with the electric saw will not always prevent injuries to the sinus and dura.

Osteoplastic is technically impossible, because, in the first place, we have to deal with a plate-like bone in the upper part of the sinus and in the dependent part of the sinus sigmoideus with the broad and

extensive mastoid process; and, in the second place, these osseous parts are purulently inflamed, and for that reason alone could not be utilized for osteoplastic purposes.

The venous sinus being sufficiently exposed, it is necessary to examine its contents. The questions to be decided are:

(1) Is there any thrombosis in the exposed area?

(2) If so, is it parietal, merely narrowing the lumen of the sinus, or is it obturating, completely eliminating the lumen at a more or less circumscribed place?

To decide these questions, it is necessary to open the sinus. Examination with the aspirating needle does not furnish a reliable and positive result, for the following reasons: If the end of the needle remains in the area of the thrombus, it will be impossible to aspirate any fluid contents, even in many cases of parietal thrombosis; when it is possible to aspirate blood, it is still doubtful whether there is a parietal thrombosis or no thrombosis at all. It is only in those rare cases where pus can be aspirated that a diagnosis of obturating thrombosis can be made.

A puncture of the exposed sinus with the scalpel, 1–2 mm. in length, will yield much better results. In normal cases the blood will be expelled in a jet; if the blood exudes slowly or appears in driblets, it is a sure sign of a stenosed lumen or parietal thrombosis. In the absence of any blood, the puncture is enlarged to 5–6 mm., and, as soon as the thrombus becomes visible, it is detached from the wall of the sinus with a blunt instrument. If there is still no blood, there is sure to be an obturating thrombosis.

The presence of the thrombus having been demonstrated, its removal follows next. Parietal thrombi can but very imperfectly be removed with instruments, if at all. The sinus puncture is prolonged to 8–10 mm. A sharp spoon is carefully introduced with its concavity toward the lateral wall of the sinus, so that, when it is withdrawn, none but thrombi attached to the wall will be removed. In obturating thrombosis the lateral wall of the sinus is dissected longitudinally in an upward direction; the thrombus is detached from the wall and its upper pointed end developed. This end usually extends far into the sinus (Fig. 105), and there is always considerable hemorrhage in fetching up this end piece. By gentle pressure with an iodoform wick carrier the hemorrhage is immediately arrested. The next step is to divide the lateral sinus wall in the direction of the lower end of the thrombus, the extraction of which is again attended with hemorrhage if the bulbus venæ jugularis still contains flowing blood. This hemorrhage is arrested by advancing an iodoform wick into the lumen of the sinus. The loss of blood will only be slight in the hands of an experienced operator, as the hemorrhages are expected after mobilization of the ends of

the thrombus and the iodoform wicks would be held in readiness. In parietal thrombosis the lateral wall of the sinus is left *in situ* after the division. In obturating thrombosis the lateral wall of the sinus is resected as far as there are any inflammatory changes, after the thrombus has been removed and the hemorrhage arrested. If the medial wall of the sinus is smooth and glistening and shows a gray discoloration, the operation on the venous sinus is at an end. The wound is covered with iodoform wicks and the skin flaps are fastened over them with a bridle suture. Should the medial wall of the sinus be dull, fragile, and of grayish-yellow or greenish color after resection of the lateral wall of the sinus, it is a positive sign of the phlebitis having spread to the medial sinus wall. This would point to the probability of the presence of pachymeningitis and encephalitis (meningo-encephalitis). In order to decide positively whether there is pachymeningitis, requiring the division of the dura and opening of the intradural spaces, a lumbar puncture must be made. Full details on this subject are contained in the chapter on pachymeningitis interna.

BULBUS THROMBOSIS

Inflammation of the bulbus of the vena jugularis may take place in three ways:

(1) By a thrombosis of the sinus sigmoideus spreading downward to the bulbus jugularis and the vena jugularis interna.

(2) By continuous or metastatic spreading of the ulcerative process through the fundus of the tympanic cavity to the vena jugularis. Direct extension of the suppuration occurs preferably in those cases where the bulbus, which is located in the fossa jugularis, is separated from the tympanic cavity by a thin bony layer, or where in consequence of osseous gaps there is no complete separation at all between the bulbus and the hypotympanum.

(3) By the extension of a pus focus in the mastoid. This occurs in cases where the fossa jugularis and the hypotympanum are separated by a bony layer which, although very broad and sometimes up to 6 mm. thick, is permeated by numerous air spaces which communicate with the air spaces of the mastoid and are promptly attacked in suppurative mastoiditis. Under such circumstances there is the same danger of infection for the bulbus which exists for the sinus sigmoideus in acute mastoiditis.

Symptoms.—Like the thrombus of the other venous sinuses, that of the bulbus is associated with clinical manifestations of otitic pyæmia. There are no positive clinical signs, however, which admit of an exact clinical diagnosis of bulbus thrombosis before the operation.

It may, however, be said in a general way that bulbus thrombosis should be thought of in cases of chronic suppuration of the middle ear

with far-advanced destruction of the middle ear, and especially in cases of cholesteatoma of the hypotympanum and chronic suppurative mastoiditis. In most chronic and all acute cases, however, the operative findings alone permit an exact decision. The diagnosis of bulbus thrombosis is very probably correct if, after exposure of the sinus, the inflammatory changes and the thrombosis are still demonstrable in that part of the sinus sigmoideus which is deflected in an anterosuperior direction. A fully developed bulbus thrombosis may be positively diagnosticated if the jugularis interna is found to be empty, or if the blood exudes from the peripheral end of the jugular with slight pressure or only in drops. The venæ condyloideæ, the sinus petrosus inferior, and sometimes the terminations of the vertebral veins inosculate below and in the neighborhood of the bulbus jugularis. If the thrombus of the bulbus has spread to the inosculations of those veins, or perhaps to the veins themselves, the jugular is entirely deprived of any blood supply, and the peripheral end of the jugular, when opened, is found to be empty. Should the inosculations be still uninvolved, then it is merely the principal arm of the affluent blood that has been injured by the bulbus thrombosis, so that the evacuation of the blood from the sinus sigmoideus into the jugularis interna is rendered difficult or impossible. In such a case there is still flowing blood in the vena jugularis interna, which, however, exudes with but slight pressure and in small quantities.

Treatment.—There are two methods of treatment: (a) the surgical exposure and opening of the bulbus, and (b) the surgical drainage of the bulbus.

Grunert exposes the bulbus after resection of the mastoid apex, free exposure of the sinus sigmoideus, and ligature of the jugular. He follows the sinus sigmoideus up to the bulbus and after detaching the surrounding muscles (sternocleidomastoid, digastricus, splenius capitis, and rectus capitis lateralis) exposes the bulbus from behind, gradually removing the osseous cover. This may necessitate the resection of the transverse process of the first cervical vertebra. According to this method the posterior surface of the bulbus presents itself first. This, however, involves the great danger of injuring the facial nerve, and, besides, a satisfactory exposure is impossible if the bulbus has developed strongly upward.

Tandler combines the retro-auricular skin incision with the ligature of the jugular, inverts the sternocleidomastoid backward, and exposes the bulbus in a perfectly satisfactory form without any danger to the accessory and facial nerves.

Piffl's method consists in exposing the bulbus, starting from the auditory canal, by removing the inferior wall of the osseous auditory canal and of the fundus of the tympanic cavity, the exposure commencing

at the head of the bulbus istelf. By further removing the soft parts and the bone, the exposure may be extended to the jugular.

Voss starts from the medial wall of the bulbus.

In order to obtain reliable drainage, the sinus sigmoideus may be followed over the lower knee anterosuperiorly up to the bulbus. By removing the wall of the sinus, the vast extensive cross-section of the sinus will admit of liberal access to the contents of the bulbus, which may now be removed along with the wall of the bulbus with the aid of the sharp spoon. This can be done without any difficulty. Reliable and sufficient drainage of the bulbus may be effected near the auricular wound by introducing iodoform wicks, while a reliable drainage of the bulbus may be effected through the jugular itself by means of the skin fistula of the jugular which I have devised. In most cases I have observed, drainage has proved satisfactory except where fetid thrombosis and suppuration of the bulbus rendered it necessary to remove completely the lateral osseous wall after resection of the mastoid. I have never found it necessary, however, to resect the transverse process of the atlas.

Clinical Course and Prognosis.—After drainage has been established, the secretion of pus from the bulbus usually continues for six to ten days. The first change of bandage should, therefore, take place as early as the second or third day after operation, and repeated daily in order to prevent retention.

The prognosis does not differ from that of otitic thrombophlebitis and will be described under that head.

LIGATION OF THE JUGULAR. SKIN FISTULA OF THE JUGULAR

In order to prevent the spreading of pus in otitic thrombophlebitis and pyæmia, Zaufal recommended, in 1889, the ligature of the vena jugularis interna of the healthy side. His idea was that this vein was the principal centripetal channel for conveying the contents of the infected auricular venous sinus, and that, consequently, its ligation was the best way to prevent spreading of pus, and that such spreading would be arrested if it had already set in. The theoretical foundation of this operation is not free from objections. Pus and bacteria may be conveyed from the auricular region by other vessels, aside from the jugularis interna, as for instance the venæ condyloideæ and the vertebral veins, the latter anastomosing in nearly all cases with the deep as well as with the superficial cervical veins. Furthermore, pus may be spread by retrograde conveyance through the sinus transversus, the sinus sigmoideus, and the jugular of the healthy side. The possibility of conveying pus in a direction contrary to that of the blood current is explained by the fact that the venous blood flows intensely only in the axial part of the vessel, while there is but slight movement in the proximity of the walls. At such

places where the vessel suddenly changes its direction (knee of the sinus, bulbus), part of the current may be completely arrested, there may be whirls, and, in some parts at least, actual reversion of the current.

It is not surprising, therefore, that the ligation of the vena jugularis interna as a preventive of metastasis is not always successful, and that metastases do often occur in spite of the ligature. Furthermore, metastases may have existed before the jugular was ligated and new metastases may develop from the old ones. The unfavorable results were further increased by the fact that Zaufal's suggestion was misunderstood. The idea was entertained that the ligation of the jugular was a sufficient surgical measure for the treatment of otitic pyæmia, so that the exposure and drainage of the auricular pus focus were either omitted altogether, not carried out in time, or done imperfectly.

In conjunction with surgical treatment of otitic pyæmia, ligation of the jugular is a very valuable and beneficial proceeding.

However, I have substituted for this method the skin fistula of the jugular, which is made either primarily at the time of operation or secondarily from one to three days after the operation. The neck of the patient is stretched by placing a small cushion underneath it and the head is turned toward the healthy side. An incision is then made on the affected side in the middle third of the neck, exposing the jugularis interna. The incision is about 4 cm. in length and is made at the anterior border of the sternocleidomastoid. After resection of the platysma and moving the musculo-cutaneous nerve to one side, the muscle is bluntly dissected free and rolled away in a postero-exterior direction. This brings into view the lateral wall of the jugularis interna, covered with a vascular sheath. The latter is either bluntly divided at its posterior circumference or longitudinally intersected over a hollow probe. The vein is now dissected free by a circular incision. Should the vena facialis communis appear in sight and interfere with convenient mobilization of the jugular, it is doubly ligated and cut through between the two ligatures. This procedure will not be necessary if the ligature of the jugular can be effected in such a way that the inosculation of the vena facialis communis will lie centrally from the ligature of the jugularis.

If flowing blood should be found in the jugularis interna, the fistula should not be made for the moment. The vessel is doubly ligated, cut through between the two ligatures, the long ligature threads being conducted outward. As a rule, the ligature at the peripheral end of the vein may be removed in twenty-four hours without any danger of hemorrhage, after which the skin fistula may be proceeded with. The ligature of the central part of the vein can be removed in from eight to ten days, provided no complications set in.

Should the vena interna be found to be empty and the wall of the vessel unchanged, it is advisable to place the fistula in the upper angle of the cervical wound, so that the natural drainage tube need not be unnecessarily long; at the same time, it will prevent stagnation of the secretion. Should the jugular contain thrombi or pus and the wall show inflammatory thickening, it will be necessary to expose the jugular further centrally, until a wall of normal appearance has been reached. This may further lead to the necessity of temporarily resecting part of the sternocleidomastoid. Should the phlebitis extend very low down, it may even be necessary temporarily to sever the muscle from its insertion at the sternum. On the other hand, I never found it necessary to resort to the temporary resection of the clavicle (Grunert). The central ligature is made as low down as possible, the peripheral lumen of the jugularis remains open, the open cross section of the jugular is enlarged by a longitudinal incision through the wall, about 1 cm. in length, and the vena jugularis is fixed by interrupted sutures in the skin wall along the more or less oval aperture which has been provided. In this way the contents of the vein are drained outward for the entire length of the peripheral part of the jugular vein, and the drainage of the bulbus is effected through the skin fistula of the jugular.

In suppurative periphlebitis of the jugular, adhesions between the vein and the surrounding connective tissue have been observed, likewise infiltration of the soft parts of the neck. In such cases extirpation of the jugularis interna (Fig. 110) and direct drainage of the bulbus are the indicated measures.

The lumen of the jugular is kept open by wicks which are inserted in an upward direction. In other respects the cervical wound may be closed.

In all cases of diagnosticated otogenic thrombophlebitis and pyæmia, I ligate the jugular as a preliminary step to the ear and sinus operation. The second step in acute cases is antrotomy and resection of the mastoid, in chronic cases the radical exposure of the middle-ear spaces. The third and last act consists in the exposure and opening of the affected venous sinus, including the bulbus if necessary, and in the removal of the thrombi.

By planning the operation in this way, the exclusion of the jugular offers great advantages. It is an improvement in the sense of Zaufal's original suggestion, because the exclusion of the largest centripetal blood canal overcomes the danger of a general infection and metastases in a large number of cases.

Brieger recommends the ligation of the jugular in cases where the pathological focus cannot be completely reached from the auricular wound, or where the sinus furnishes negative findings and the existing

pyæmia must necessarily be referred to thrombosis of the jugular. Kuemmel believes that the manipulations necessary in ligating the jugular may dislodge parts of the thrombus in the jugular and may be the cause of pulmonary metastases.

Ligation of the jugular as a preliminary step to operations on the ear and sinus protects against the danger of parts of the infected thrombus being mobilized and carried into the circulation during the act of removing the bone which involves an unavoidable trauma of the sinus.

If the diagnosis of thrombosis is only made at the operation, the latter is suspended until the jugular has been ligated. This arrangement makes provision by a single operation for everything that is necessary for the surgical treatment and healing of otitic pyæmia. The ligation of the jugular, the skin fistula of the jugular, the continued exposure of the affected sinus until healthy parts have been reached, the timely opening of the venous sinus, and the removal of the thrombotic masses, protect the patient from the danger of having to undergo several or renewed operations in case the original incomplete proceeding (insufficient exposure of sinus, impractical exposure of the affected parts, omission of excluding the jugular) should not meet the requirements of the case. Otogenic pyæmia causes exceedingly rapid degeneration of the heart muscle and the large glands. Each fresh anæsthesia, each fresh operation, favors the progress of the degeneration, aside from the fact that subsequent completion of the operative measures is too late in most cases and unable to prevent a fatal issue.

It is admitted that a mastoid operation may be satisfactory where there is no highly virulent infection and in cases of sterile thrombosis, but it is impossible to determine the virulence of the pyogenic factors or the infectious properties of the thrombus before operation. The idea of doing a simple operation and resorting to exposure and opening of the sinus and ligation of the jugular vein, in case the original operation should prove insufficient, is a mistake, inasmuch as it will only answer in a small number of cases of slightly infected or sterile thrombi the outcome of which will depend upon a mere accident. In most cases this waiting attitude or "fractional operating" will prove disastrous, because subsequent operations which may be required by persisting pyæmia are usually in vain. The extent to which the operative findings can influence our decision is limited to the question of the size of the exposure of the sinus and bulbus regions.

PYÆMIC METASTASES

Otitic phlebothrombosis may be the starting-point of otitic pyæmia, bacteræmia or toxæmia. Pyæmia will occur from pus and micro-organisms entering the circulation from the original otogenic focus in the

venous sinus, causing suppurative metastatic inflammation by infectious deposits in various parts of the body.

According to the localization of the metastases, the following forms of otitic pyæmia have to be distinguished: (1) the cranial form, (2) the thoracic form, (3) the abdominal form, (4) metastases in the bones, muscles, and joints.

Of course, there are often mixed forms, but clinical experience causes us to adopt the above division, the more so as it is also of importance for the prognosis.

In *cranial otitic pyæmia* there are metastases in the bone itself, pyæmic cerebral abscesses, pachyleptomeningitis or pachymeningitis interna, metastatic suppuration of the cranial bones, cavities of the eye, or accessory cavities of the nose. Generally speaking, this form of pyæmia is rare. The cases of cerebral abscess which have been observed in otitic pyæmia conform to the interpretation that the abscess has not occurred by metastasis, but by direct spreading of the pus focus from the venous sinus to the endocranium.

Pachyleptomeningitis in cases of otitic pyæmia is, unfortunately, not a rarity. In one-half of all fatal cases due to otogenic pyæmia the cause of death is suppurative meningitis. A small number of these are of metastatic origin, but the majority occur by direct spreading or perforation of the endosinous pus focus. On the other hand, a small number of positive cases of metastatic pachymeningitis interna have been reported, in which the inflammatory foci of the endodural surface must be looked upon as metastases of the pus focus in the sinus.

The *thoracic form of pyæmia* is characterized by the metastatic pulmonary abscess. This form of otitic pyæmia is of frequent occurrence. There may be one single abscess, but in many cases there are a large number of them. Both lungs may be attacked by the suppuration. The smaller bronchi as well as the larger bronchi of the affected pulmonary region will also be invaded in a relatively short time. With perforation of the abscess there will be suppurative pleurisy, suppurative bronchitis, and empyema of the lungs. Metastatic inflammation of the heart and pericardium are by no means rare in long-persisting pyæmia.

Pyæmic metastases in the kidneys are also rather frequently observed, while abscesses in the liver and spleen as well as metastatic suppurative peritonitis or cystitis are very rare.

Muscle metastases are oftener found in the regions of the neck, shoulders, and buttocks than in other parts of the body. I have often observed large abscesses of the muscles in the region of the scapula, especially the deltoid, but of course abscesses may occur in any other muscle. In most cases the muscle abscesses commence after the type of phlegmons, suppurative disintegration starting in the muscle sub-

stance itself. If the pus has perforated through the fascia, widely ramified descending or multiple abscesses may develop. I once saw a complicated abscess formation of the perineum which had started from a suppurative otitic metastasis of the glutæus medius muscle.

Abscesses of the muscles of the abdomen and extremities usually remain well localized, and are soon recognized by the circumscribed swelling and reddening of the skin.

Concerning *suppurative metastases in the bones*, the femur, scapula, tarsus, humerus, carpus, and the phalanges may be mentioned. In one case I observed suppurative metastasis in the ribs, sternum, and third cervical vertebra. Suppurative inflammation of the joints themselves is fortunately rare. I observed a case in which the metastasis ran a five weeks' course, starting with an extensive metastasis of the left knee-joint and spreading to the right knee-joint, the right astragalus, the right carpus, later to the left carpus, and finally to the sternoclavicular articulation.

A ten-year-old boy was taken ill with otitic otogenic pyæmia following an acute scarlatinal otitis. A metastatic coxitis developed in the right hip-joint, which terminated with ankylosis and shortening of the leg. In the course of the disease there developed an osteomyelitic abscess in the upper part of the femur of the affected side and in the large trochanter.

Symptoms and Course of Metastases

Symptoms.—The formation of metastases is principally recognized by the continuance of the pyæmic general manifestations (intermittent fever, chills, icterus) after the pus foci of the ear and venous sinus have been surgically removed. Local pains often point to the place of the metastatic inflammation, but patients are liable to commit considerable errors in their localization. Thus, the patient mentioned above, suffering from metastatic coxitis, always referred his pain to the lower end of the femur, and it required a thorough radiographic examination to clear up the real seat of the disease. Metastases in the muscles are more correctly localized; here, again, the seat of the inflammation is usually recognized without any difficulty. Should there be pain in the orbit, it is of the utmost importance to test carefully the functional capacity of the eye muscles.

Metastases in the accessory cavities of the nose may begin and end without causing any pain. The suspicion of their presence is aroused by acute occlusion of one side of the nose, due to swelling of the lower or middle turbinated bone or by spontaneous secretions. The situation is cleared up by a thorough examination of the nose and its accessory cavities.

Pulmonary abscesses, too, may exist without giving rise to any symptoms, as they may only cause trouble after pleurisy has developed

in the region of the abscess. Irregularities of respiration demand careful examination of the lungs. Suppurative abscesses which are situated near the surface of the lungs cause the thorax of the affected side to remain distinctly behind in the act of respiration.

Abdominal metastases are recognized by the distention of the abdomen. There is tension and hardness of the abdominal wall, and often there is retention of urine at an early stage.

X-ray examination for establishing and localizing pyæmic metastases cannot be too urgently recommended.

Diagnosis.—The diagnosis of metastases in the muscles and joints usually presents no difficulties. Their presence is indicated by local pain and the continuance of pyæmic manifestations. Muscle abscesses can be palpated without great difficulty. In order to diagnose metastases in the articulations and long tubular bones, it is sometimes necessary to make repeated X-ray examinations. As to the diagnosis of metastases of the thoracic and abdominal viscera, the text-books of internal medicine should be consulted.

If the changes of the ear itself and the regional pathologic manifestations should not sufficiently explain the continuance of the pyæmic conditions, it will not do to be content with the vague assumption of the presence of pyæmia. Metastases should be energetically looked for at an early stage, not forgetting the infrequent ones (spleen, liver, peritonitis and cystitis, etc.).

Treatment.—In young patients, and especially in children, there is a favorable possibility of suppurative metastases completely healing without operation. I saw an ideal recovery from a metastatic endo- and pericarditis in a boy of four years; in a girl eight years of age there was spontaneous involution of a pulmonary abscess,—that is, healing of a pulmonary infarction.

Among the internal remedies the following may be mentioned: Nucleic acid (5–20 c.c. subcutaneously), nucleic sodium and nucleogen—Rosenberg (2 or 3 tablets daily), argentum colloidale and electrargol (5–20 c.c. intravenously or per rectum). In the period of high fever, cold packs are indicated; liberal fluid nutrition (if necessary by nourishing enemas) and inunction with unguentum Credé will be useful. In cases of a septic character, tentative injections of streptococcus serum and autovaccination are justified. The streptococcus injection, however, is only applicable if streptococcus infection has been demonstrated, 1–4 injections of 5–10 c.c. each being made at intervals of 2–3 days.

It is always advisable to open the metastatic focus as early as possible, except in metastatic osteomyelitis, where the full development of the abscess should be awaited; otherwise it might become necessary to

resort to an unnecessarily extensive exposure of the marrow in the long tubular bones. It might also happen that, in spite of extensive evacuation of a part of the bone, a second or even third operation will become necessary owing to the continuance of fever and pain. In metastatic abscesses of the knee-joint, repeated puncture of the joint usually leads to complete recovery with good motility. Metastatic coxitis, however, always leads to ankylosis.

Course of Temperature in Otitic Pyæmia

Otitic pyæmia is characterized by intermittent fever. In thoroughly characteristic cases the rise of temperature from normal or subnormal to very high degrees of fever occurs in a very short time. The high temperature is maintained for some time and recedes shortly before a fresh accession takes place. In many cases there is only one accession in 24 hours, in others there are several in the same time. It may be said in a general way that the number of accessions increases with that of the chills. In some cases there is only one characteristic chill at the beginning of the pyæmia, which is suddenly followed by fever temperature. In these cases the intermittent type of the fever is not very distinctly apparent. There are neither declines to the normal nor very high elevations. It may be said, from the study of a large number of temperature curves, that there is already ground for suspicion of otogenic pyæmia in cases where the lowest temperature in the course of a day is 97.7° or less, and the highest 100.95° to 101.5°, amounting to a difference of at least 2.25°. It is a dangerous symptom for the intermittent type to change to the continuous in the course of the disease, as the occurrence of continually high fever following in the wake of intermittent fever is to be looked upon as an indication that direct spreading of pus has led to a metastatic suppurative meningitis.

Continuous fever lasting for a day or more at moderate height or normal temperature may be followed by a fresh rise of temperature caused by the development of a fresh metastasis. After recovery has taken place, intermittent fever usually occurs for a few days longer than the local pathological manifestations.

Postoperative fever in otogenic pyæmia deserves particular attention. The most favorable course is decrease of fever by lysis in such a way that, on the fifth or sixth day after operation, the temperature will be normal and remain so. With the occurrence of new metastases after operation, intermittent fever will of course continue, and on the occurrence of new chills there will be characteristic and important variations in temperature.

Return of temperature to normal immediately after the operation admits only exceptionally of a favorable interpretation, and that is

when the pyæmic process is at once arrested and cured with the operation. In that case the temperature may at once return to normal and remain so. In most cases, however, where normal temperature returns immediately after the operation or on the following day, it means a collapse temperature and is to be looked upon as unfavorable. It is explained by a further loss of strength the patient has undergone from the operation, and an appreciable rise will occur in the next two or three days; and on the fifth or sixth day, at a time when in favorable cases normal temperature is being attained, high pyæmic fever accessions will again prevail. In unfavorable cases the intermittent type will sooner or later change into continuous fever. In cases with fatal termination there is usually a sudden decline of temperature shortly before death, associated with an exceptionally high pulse frequency.

Pulse and respiration are accelerated in all cases of otogenic pyæmia, 160–180 pulse-beats being no rarity in young patients. The pulse acceleration is often still present for several weeks after the pyæmia has run its course.

It is important to observe the respiration, because its abnormal behavior is often a sign of a pulmonary abscess. As to the details of this phase the text-books of internal medicine should be consulted.

Postoperative Prognosis of Otogenic Pyæmia

The pathological material, consisting of 96 cases which I have observed and operated upon, shows a mortality of 16 per cent.

The mortality of otogenic pyæmia is, therefore, higher than that of extradural abscess and chronic suppuration of the labyrinth. It is lower than in acute diffuse suppuration of the labyrinth, abscess of the temporal lobe or cerebellum, or infectious suppurative meningitis. Comparison of the present mortality of 16 per cent. with that of 80–90 per cent. in the pre-operative era will show the advantages afforded by exact indications and systematic planning of the operation.

Will it be possible to reduce the mortality still further?

To decide this question it is necessary to study the autopsy findings for the changes from which the unfavorable cases died.

Some eight years ago I compiled the autopsy findings on the occasion of a comprehensive paper, showing the following causes of death:

(1) Local changes of the ear in cases where the pus focus in the ear and venous sinus failed to heal. The principal contributors to this group are the formerly very frequent cases in which otitic pyæmia and thrombophlebitis were not accurately diagnosed and consequently could not be adequately and promptly operated upon.

(2) Pyæmia or sepsis with far-advanced degeneration of the pericardium and the large glands (spleen, liver, kidney).

(3) Non-diagnosed metastases which had led to fatal affections.

(4) Infectious suppurative meningitis and acute encephalitis.

The second group comprises all those cases in which pyæmia had run a very chronic course and there was the necessity of repeated operative interference; furthermore, those cases in which the affection showed rather a septic character or where the power of resistance had been lowered by other affections.

The third group contains cases with suppurative metastases in unusual situations which could not be easily traced (spleen, liver, and kidney abscesses with purulent pyelonephritis). In these cases there is the danger of the metastases remaining undiscovered for a long time, until the abscesses perforate, causing death by extensive suppurative disintegration.

The percentage of cases which are complicated by suppurative pachyleptomeningitis is still considerable to this day. In spite of timely diagnosis, exact indication, and efficient operation, we are in a certain number of cases unable to prevent the development of meningitis.

With these considerations it is possible to furnish a fairly clear answer to the question as to whether we may be able still further to reduce the mortality of these cases. Exact clinical examination, leading to a correct diagnosis at an early stage, is of great importance. If, based upon this diagnosis, operation is performed sufficiently early, and to a sufficient extent from the first, there is no doubt that the percentage of cured cases will be raised. The number of those cases in which patients have succumbed to degeneration of the pericardium and the large glands, owing to repeated operative measures, will be reduced by planning the operation in a systematic manner and carrying it out at one sitting.

III. OTITIC SEROUS MENINGITIS

Serous meningitis occurs more frequently in acute inflammation of the middle ear or labyrinth than in chronic cases. Many of them present a definite pathological picture, others resemble the early stages of acute suppurative or tuberculous meningitis. The affection is far more frequent in children than in adults.

Anatomy.—The principal sign is the inflammatory increase of the cerebrospinal fluid with consequent elevation of the intracranial pressure. The meninges are hyperæmic, highly osmotic, and in long-existing cases gray or grayish-white discolored in circumscribed places. There is in many cases early œdematous swelling of the cerebral surface leading to acute encephalitis. The changes usually extend over the entire cerebral surface, often involving the spinal meninges. It is only rarely that the meningitic changes remain circumscribed and in close topical relation to the affected ear, but it is just in these cases that there is usually serous

meningo-encephalitis as a preliminary stage to acute suppurative or tuberculous meningitis.

Complete healing occurs in uncomplicated cases. The quantity of cerebrospinal fluid is reduced to normal, and the inflammatory manifestations of the cerebral meninges and of the brain completely disappear. If serous meningitis is the precursor of purulent meningitis, the suppuration will follow the acute stage either immediately or only after a prolonged existence of the original affection.

Cerebral manifestations capable of simulating meningitis may occur at the onset of acute otitis media previous to the perforation of a suppurative exudate through the tympanic membrane. This symptom, for which the term "meningismus" has been proposed, will disappear as soon as the pus secretion from the middle ear commences after paracentesis.

Symptoms.—The principal symptom of serous meningitis consists in early delirium, disturbance or loss of consciousness. The latter may persist for days. As a rule, there are early lagophthalmos and moderate rigidity of the neck. The fundus of the eye is unchanged. Should there be pathological changes, we have to deal not with serous meningitis proper, but with a serous preliminary stage of suppurative meningitis. Vomiting in serous meningitis is rare; slow pulse will persist for a long time.

It is a curious fact that nutrition of the patient will succeed fairly well by careful introduction of fluid food, in spite of disturbed consciousness. At the climax of manifestations there may be incontinence of urine and fæces. There are no convulsions or paralysis of the extremities. The latter are relaxed, and the motor innervation is weakened, but not entirely suspended.

Diagnosis.—The exact diagnosis and differentiation of serous meningitis as against the other forms of meningitis follow from the symptoms as explained above and from the findings of lumbar puncture (see p. 355). The cerebrospinal fluid flows off under increased pressure and is clear and sterile both microscopically and in cultures. Standing undisturbed in a test-tube for six to twenty-four hours will not cause any coagulation.

The course of serous otitic meningitis is nearly always favorable. The manifestations gradually diminish, and upon return of consciousness there is rapid recovery.

The question of **treatment** of serous otitic meningitis is not yet quite settled. However, immediate energetic evacuation of the purulent exudate in the middle-ear spaces is unquestionably indicated at the onset of meningitic manifestations. With a still imperforate tympanic membrane, free paracentesis will often be attended with good

results. Should the manifestations show no abatement in the course of twenty-four hours, antrotomy and lumbar puncture are indicated in acute suppuration of the middle ear. In chronic cases lumbar puncture must be preceded by the radical operation. Opening of the dura is only indicated if the local suppurative inflammation of the temporal bone can be continuously followed up to the dura and the external surface of the dura shows inflammatory changes. In the absence of circumscribed changes the mere exposure of the dura will suffice without incision. The first change of bandage is made twenty-four hours after operation, when in favorable cases the tension of the dura will already be diminished.

Should increased tension persist in spite of lumbar puncture, a longitudinal incision is made into the dura, taking care not to injure any of the larger dural vessels. As a rule, the œdematous brain will at once protrude into the incision, so that the otherwise desirable insertion of drainage strips into the intradural space will succeed only in exceptional cases. The dura being incised, any further lumbar punctures should be avoided. If the dura is not incised, lumbar puncture should be repeated at intervals of five days, evacuating on each occasion 5–10 c.c., or, in cases of considerably exaggerated pressure, up to 20 c.c.

Attentive nursing is of the utmost importance in serous meningitis, in connection with which it is important to see that, in spite of disturbed consciousness, liquid food (milk) should be ingested and retained. By careful closure of the lids and laying moist compresses upon the eyes, acute keratitis due to lagophthalmos should be prevented. The skin should receive due care in order to prevent decubitus during the period of disturbed consciousness.

After the serous meningitis has been cured, relapses need not be apprehended, children particularly recovering very rapidly without showing any impairment of their intellectual qualities.

IV. CIRCUMSCRIBED SUPPURATIVE PACHYLEPTOMENINGITIS AND INTRAMENINGEAL ABSCESS

Anatomy.—Circumscribed otitic meningitis usually starts from inflammatory foci located on the medial surface of the dura. In the region of these foci the latter is thickened, grayish yellow, yellow, or yellowish green discolored, and dull. There are pus deposits in advanced cases, causing comparatively early infection of the leptomeninges and encephalitis. The anatomical course varies. There may be agglutination of the pachy- and leptomeninges in the inflamed region, followed by regional adhesions, so that the spreading of pus and diffuse meningitis need not be apprehended. In other cases an intrameningeal abscess will develop, which may perforate and lead to a dural fistula and extradural abscess. In unfavorable cases the intrameningeal abscess will spread further,

leading to cerebral abscess or diffuse purulent meningitis, either by direct continuity of the inflammation or by way of metastasis.

Another contingency is the formation of descending endocranial abscesses in cases where the pus accumulates at the lowest point of the medial or posterior cranial fossa.

Etiology.—Circumscribed pachyleptomeningitis is usually caused by chronic middle-ear suppuration, complicated by an affection of the dura or a cerebral abscess. There is also danger of an intrameningeal abscess in many cases of long-persisting purulent sinus-phlebitis or of pachymeningitis externa. Circumscribed pachyleptomeningitis with cerebral abscess will occur at the place where the abscess is preparing to perforate. In this respect it is not an unfavorable complication, provided the intradural space is obliterated by inflammatory agglutinations in the region of the perforation, so that there is no apparent danger that the intact part of the intradural space will be invaded by the escaping pus. An intrameningeal otitic abscess will sometimes occur in the basal part of the posterior cranial fossa in chronic suppuration of the labyrinth or sinus thrombosis.

Circumscribed pachyleptomeningitis is very rare in acute middle-ear suppuration, and nearly always occurs by metastasis on the basis of acute, progressive extradural abscesses. It may also occur in the course of malignant tumors of the ear after exulceration, such tumor having either spread to the dura in the course of growth or having originated in the dura. Furthermore, circumscribed meningitis is observed in fractures or fissures of the temporal bone if at the time of injury, or later, the middle ear has become infected, causing otitis media.

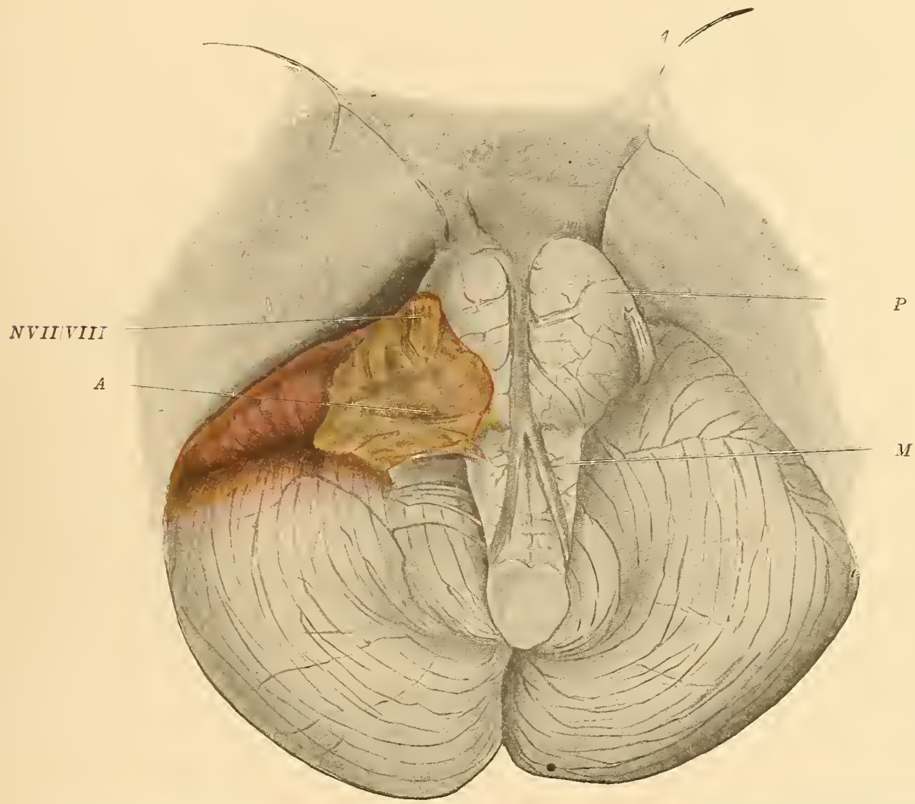
Subarachnoid abscesses of the posterior cranial fossa are sometimes caused by chronic suppuration of the labyrinth, with spreading of the pus to the internal auditory canal, and by suppurative otitis of the petrous bone (Fig. 112). These deep abscesses are an exceedingly dangerous complication of labyrinth suppuration.

Symptoms.—Circumscribed pachyleptomeningitis is accompanied by stinging headache, referred to definite places, which, however, do not always correspond to the seat of inflammation. With extension of the affection to the parietal lobe, considerable accumulation of exudate may cause motor disturbances by impairment of the motor cortical region (spasms and, later, paralysis). In most cases there is sensitiveness to percussion of the temporal squama. Circumscribed pachyleptomeningitis of the posterior cranial fossa is accompanied from the onset by rigidity of the neck. Opisthotonos is usually present as soon as the meningitic changes extend to the base of the skull. The fundus of the eye shows unilateral or bilateral venous plethora. The cerebrospinal fluid is gray, turbid, and contains abundant mononuclear and poly-

nuclear leucocytes; in most cases it is sterile. Aërobic or anaërobic micro-organisms of the most varied kinds are found in the meningitic foci. Proteus is found only exceptionally.

Diagnosis.—It is only exceptionally that the clinical diagnosis of circumscribed meningitis can be made with absolute certainty, as in most cases the symptoms of the causative affection preponderate (sinus

Fig. 112.



Cerebral base of a boy, fourteen years old. Subarachnoidal abscess (*A*) and circumscribed leptomeningitis (red) of the right cerebellar hemisphere in the area of the insertion of the auditory and facial nerves (N. VII, VIII). Natural size. *P*, pons; *M*, medulla oblongata.

thrombosis, suppuration of the labyrinth, cerebral abscess, etc.). It is absolutely impossible to recognize the early stages of pachyleptomeningitis, as they may run a course entirely devoid of symptoms. At the time of operation a dural fistula, if present, would certainly disclose an intrameningeal abscess, but fistula formation is a comparatively rare occurrence. It follows that in an endocranial affection of the external dura (pachymeningitis externa, sinus thrombosis) we are, even with the advantages afforded by an operation, dependent upon other signs which render the presence of an intrameningeal abscess probable. These include considerable swelling of the dura, which is deeply loosened, in

other cases red or yellowish-green discoloration, fragility, inordinately strong tension or flexibility of the dura, deep fetid pus foci of the dura in sinus thrombosis, and also yellowish-green discoloration and fragility of the medial sinus wall, which can be inspected after opening the sinus and evacuating the thrombus. An intrameningeal abscess will betray itself in cases of labyrinth suppuration by the fact that after exposure of the dura, when resecting the labyrinth, pus will protrude from the region of the saccus endolymphaticus or from the apical region of the semicircular canal or from the internal auditory canal.

An intradural abscess cannot escape attention in cases of abscess of the temporal lobe or cerebellum, since it is necessary anyway to open the intradural space in order to evacuate the cerebral abscess. By observing the fundamental rule in operating upon a cerebral abscess, to follow the tracts over which the ear affection has spread into the cranium, the intrameningeal pus focus is sure to be discovered.

It is just in these cases of cerebral abscess, and in many cases of sinus thrombosis, that valuable assistance will be given by the findings of lumbar puncture. Turbid, sterile fluid is of immense importance for the diagnosis of circumscribed meningitis.

Treatment.—Treatment can only be surgical. It consists in antrotomy in acute middle-ear suppuration or radical operation in chronic cases, in exposure of the dura beyond the affected area, and in free incision of the dura. The method of effecting the latter is by incising layer by layer, because in this way it is possible to observe whether the pus emanates from the extradural or intradural space or from the brain itself. Direct puncture of the dura with the scalpel or, worse, with a hollow needle may infect the brain which may have been intact before, and, besides, will give no indication as to which spaces the evacuated matter originates from (extradural, intradural, or intracerebral).

The wound is drained with iodoform or isoform wicks. The first change of bandage should take place two days after operation.

Course and Result.—Many cases of circumscribed meningitis have a distinct tendency to remain circumscribed, a clinically important fact to which Voss first called attention. The prognosis is also favorable in cases where the intrameningeal abscess has existed for some time and is walled up by adhesions against the rest of the intradural spaces. The prognosis is less favorable in cases with symptoms of general meningitis and intradural abscess of the posterior fossa. In the latter cases the prognosis is unfavorably influenced by the fact that access to the abscess can be obtained only by an extensive and serious bone operation (exposure of the sinus or resection of the labyrinth) and that the unavoidable concussion of the endocranium caused by the chisel-work involves the danger of spreading the pus and advancing the meningitis.

Another point is that there is a tendency for the pus to descend to the fundus of the posterior cranial fossa. For this reason it is urgently necessary to renew the bandage and drains frequently, in order to prevent the retention of secretion. There should be no lumbar puncture while the wound is under treatment.

V. ACUTE DIFFUSE SUPPURATIVE OTOGENIC PACHYLEPTOMENINGITIS (MENINGO-ENCEPHALITIS)

Anatomy.—In diffuse suppurative otitic meningitis there is extensive intradural and subarachnoid accumulation of pus. In many cases of fully developed meningitis the otogenic origin is still distinctly recognizable by the fact that the inflammatory manifestations of the auricular region of the middle or posterior cranial fossa are in the most advanced stage, containing the oldest changes. In other cases the meningitic changes present no difference.

These changes may spread to the spinal space in a short time. If the pus accumulation is extensive, intradural agglutinations may develop in the presence of thick, viscid pus, leading to hydrocephalus internus, possibly with occlusion of the foramen of Magendie, pus accumulation in the sinuses, at the fissura transversa, and copious accumulation of pus at the falx cerebri and the tentorium.

Every case of diffuse pachyleptomeningitis is associated from the first with superficial encephalitis and superficial cerebral œdema. There will be swelling and softening of the brain, later inflammatory infiltration, and, should the case be persistent, ulceration of the cerebral surface and abscess formation. Should meningitis have developed on the basis of a suppuration of the temporal bone extending to the endocranium, perforation may establish a primary or secondary communication between the pus focus of the extradural space and the intradural inflammatory focus.

The anatomical changes in acute meningitis increase in extent and intensity rapidly and uniformly; at times, however, there is a transitory arrest of the process, from which the clinical type of intermittent meningitis develops. It should be admitted, however, that in the unquestionably rare cases of intermittent meningitis there is usually the serous or tuberculous form at the bottom of the trouble, and but seldom the typical acute suppurative diffuse form.

The bacterial findings reveal different micro-organisms according to the individual case. In the majority of them there is but one single species of micro-organisms (streptococcus, pneumococcus, pneumonia bacillus, staphylococcus, etc.), for the reason that but one bacillus has led to the inflammation or that the predominating one has outgrown the others.

Etiology.—Otitic diffuse suppurative meningitis occurs in the course of both acute and chronic middle-ear suppuration. In the first ten years of life otitic meningitis occurs far more frequently than any other otogenic affection. This is explained by the fact that a suppurative process occurring in the feeble structure of the infantile temporal bone rapidly penetrates to the dura, whence it readily spreads to the endocranium, owing to the intimate connection between the cerebral meninges and the surface of the bone.

The impression is created that meningitis in these cases runs such a rapid course of development and termination that there is no time for other cranial affections to establish themselves, notably sinus-phlebitis or cerebral abscess. However, it is quite possible for diffuse suppurative meningitis to develop on the basis of any otitic endocranial affection.

The danger of meningitis is particularly present in acute or chronic suppuration of the upper tympanic space and the antrum. Defective drainage with consequent retention of secretion in the middle ear causes the pus to be retained at the tegmen tympani in chronic cases; with a still passable fissura tympano-squamosa there will be purulent inflammation of the dura filling out the fissure, leading to suppurative destruction of the tegmen tympani with fistula formation, and the meningitis will then start from the pus focus in the bone.

In acute otitis media, meningitis occurs most frequently in the metastatic form. There is no need for the bone to be diseased up to the dura; this is demonstrated at operation by the fact that there is no recognizable material topographic relation between the meningitic foci and the ear.

Symptoms.—The ear symptoms usually correspond to those of a middle-ear suppuration with special participation of the upper space of the tympanic cavity and antrum or with retention in the attic and antrum.

The first group also embraces cases where the communication between meso- and epitympanum was reduced or obliterated before the suppurative otitis media was established by catarrhal affections, with the result that the pus originating from a later epitympanic inflammation in the attic and antrum can only escape to the mesotympanum under difficulties, if at all. This is clinically evidenced by the fact that with all the signs of grave middle-ear infection (great pain, difficulty of hearing, fever) even free paracentesis has no effect, or the pus secretion will soon be arrested again. After a short time the posterior upper wall of the auditory canal will descend, a diffuse flesh-like thickening of the tympanic membrane will develop, the hearing acuity will be considerably reduced, and in some cases there will be spontaneous nystagmus and susceptibility to percussion of the temporal squama.

Typical mastoid symptoms are present in many cases, especially at the onset of the affection; but it should be particularly emphasized that, in spite of imminent or developed meningitis, such symptoms may be absent. Generally speaking, the cerebral symptoms and general manifestations predominate over the ear symptoms.

Cerebral and General Symptoms.—The early symptoms include headache, restlessness, and sleeplessness. Sleep, if any, will last only a short time; the patient is restless and cries out repeatedly while asleep. The lid closure is imperfect and there is involuntary twitching in the region of the facial muscles. There is either absolute or relative slow or irregular pulse, with high fever and considerably increased frequency of respiration, hyperæsthesia of the skin, paræsthesia of the extremities, and exaggeration of the tendon reflexes. Kernig's symptom (flexural contraction of the knee-joint on flexure of the leg) is positive. Free motility of the head is soon impaired, followed or not followed by rigidity of the neck. Later there will be attacks of delirium lasting a considerable time, convulsions, and typical cerebral vomiting (by the "mouthful," without any particular exertion or any subsequent feeling of relief). The pupils are narrow or unequal at an early stage and there is diminished or irregular reaction of the pupils to light. At a later stage there are often exophthalmos and spontaneous coarse nystagmus, which may be straight or horizontal. In advanced cases there are abducent paralysis of vision, disturbances of speech and respiration, and loss of consciousness. The veins of the ocular fundus are very plethoric, or there is choked disk (which, however, in rapidly fatal cases has no time to develop); finally, there is paralysis of the special senses of smell and taste and of the tongue muscles.

As the process advances there will be paralysis of the intestine and bladder, involuntary defecation and micturition, or retention of urine. There is a striking pallor of the skin both on the face and over the body. The cheeks are often tinged a deep red, which sharply contrasts against the pallor or subicteric discoloration of the surrounding skin.

Diagnosis.—The diagnosis of otitic meningitis, when fully developed, presents no difficulties on taking the above symptoms into consideration. Recognition of the early stages of the affection presupposes considerable clinical experience. Any case of otitis media with continuous fever, severe diffuse headache, restlessness, dyspnœa, and sleeplessness must arouse suspicion if the local inflammation does not run a satisfactory course. Hyperæsthesia of the skin, dermographia, exaggerated tendon reflex, and positive Kernig point to meningitis. Observation of the patient during the night is of great importance. Crying out or frequently starting up while asleep, involuntary twitching of the mimic musculature, and lagophthalmos must arouse the suspicion of

meningitis. In order to make a correct diagnosis, lumbar puncture is necessary in all such cases; in the early stages, or when the affection runs an insidious course, it will guard against diagnostic errors.

Differential Diagnosis.—Differentiation between serous and diffuse suppurative meningitis is often an easy matter. Rapid development of the pathological picture and rapid involution of the manifestations point to the serous form. Rigidity of the neck, spontaneous nystagmus, persistent high fever, slow but steady increase in the intensity of the pathological manifestations, and paralysis of the intestine and bladder point to the suppurative form. However, there are many cases in which an exact differentiation between these two forms is only possible by lumbar puncture. Should the communication between the intradural spaces and the ventricles be obliterated, lumbar puncture may be negative, as this condition makes it impossible for the cerebrospinal fluid to be evacuated.

The differential diagnosis between acute purulent and tuberculous meningitis will not cause any difficulties. Tuberculous meningitis often develops with normal or slightly elevated temperature; a higher accession of temperature does not usually occur except shortly before death. Besides, in tuberculous meningitis there are practically no irritative symptoms, such as delirium, convulsions, and unrest. In doubtful cases lumbar puncture cannot be dispensed with.

Treatment.—Diffuse suppurative acute meningitis can only be treated by surgery, commencing with the ear operation. In acute middle-ear suppuration the cerebral operation has to be preceded by antrotomy, in chronic cases by the radical operation. Instead of a chisel, Luer's forceps should always be used wherever possible. The saw is not to be recommended, as the bone dust interferes with the control of the work. The ear operation is followed by exposure of both cranial fossæ. The middle cranial fossa is opened by an incision in the shape of a cross, after which drainage wicks are inserted between the protruding brain and the inner surface of the dura. Should the dura present any pathological changes, the dura of the posterior cranial fossa need not yet be resected. In the absence of any particular inflammatory manifestations, it is advisable to resect at once the posterior cranial fossa both in front and behind the sinus sigmoideus. In the event of there being still another endocranial affection, the operation should include this as well.

Lumbar puncture is best carried out following the ear operation. The quantity to be withdrawn depends upon the pressure under which the cerebrospinal fluid is evacuated, but more than 10–15 c.c. should never be withdrawn at one sitting. If lumbar puncture were carried out preceding the ear operation, or if the quantity withdrawn be too large, there would be danger of intradural hemorrhages during the ear operation.

The first change of bandage is made two days after the operation, special care being taken to maintain intradural drainage. Unfortunately, this is not always an easy matter in pronounced cerebral œdema and superficial softening of the cerebral substance. In small openings of the dura there is danger of retention; in large openings there may be an extensive prolapse. In some cases aspiration is successful, as recommended by Manasse. From a therapeutic stand-point repetition of lumbar puncture once or twice a week may be beneficial.

Course.—In spite of the great strides made in otological surgery, the operative success in diffuse suppurative meningitis is very slight. There is some chance for the following forms:

(1) Staphylococcus meningitis, several successful cases having been reported in the literature. It is not impossible, however, that some of these cases were serous meningitis, and that the staphylococcus found at the bacteriological examination was an accidental impurity.

(2) Labyrinthogenic meningitis. This group includes cases of streptococcus and pneumococcus meningitis. It is perfectly true that labyrinthogenic meningitis properly belongs to circumscribed meningitis, and that the posterior cranial fossa is completely or exclusively attacked on the side of the affected labyrinth and close to the latter. This explains why a timely resection of the labyrinth in these cases, followed by opening of the posterior cranial fossa, will lead to a cure.

All the other forms of diffuse suppurative acute meningitis are prognostically very unfavorable. Whatever hope is entertained to effect a cure by timely and extensive operation and drainage is in a large number of cases doomed to disappointment. The thick, viscid pus accumulates in the deep subarachnoid fissures and cisterns to such an extent that there can practically be no question of effective draining or removal of the matter in fully developed cases.

VI. OTOGENIC TUBERCULOUS MENINGITIS

Anatomy and Etiology.—Tuberculous otogenic meningitis always occurs in the wake of a chronic, usually bilateral, suppuration of the middle ear. In most cases this suppuration is tuberculous, but the fact has been established that tuberculous meningitis may be caused by non-tuberculous suppuration of the middle ear in a patient suffering from a chronic tuberculous affection at some other part of the body, such as the bones, lungs, or abdomen.

The danger of contracting otogenic tuberculous meningitis is particularly great in neglected tuberculous suppuration of the middle ear, as well as in cases where the tuberculous ear affection is complicated by a tuberculous affection at some other part of the body, such as the lymphatic glands, lungs, intestinal tract, bones, and joints. Tuberculous

meningitis may also be postoperative in chronic tuberculous suppuration of the middle ear if the radical operation has become necessary. Furthermore, the danger of tuberculous meningitis exists in chronic tuberculous suppuration of the labyrinth and in tuberculous caries of the petrous bone.

The anatomical findings are characterized by the presence of miliary nodules of the leptomeninx along the blood-vessels of the brain. Extensive superficial pus foci are comparatively rare. Single or multiple cerebral tubercles will develop in many cases. The inflammatory exudate, which is of a serofibrinous or gelatinous consistency, is chiefly located at the base of the skull in the region of the large sinus, leading in a short time to hydrocephalus internus and ependymitis granulosa.

In exceptional cases a special topographical relation between the localization of the nodules and the affected ear can be recognized; usually, however, the meninges of both sides are affected in cases where only one ear is diseased and without any recognizable causal relation.

Symptoms.—As compared with acute suppurative meningitis, the tuberculous form is characterized by its insidious development and but slight intensity of whatever symptoms may occur. It remains in a latent condition for a long time. The prodromal manifestations consist in a gradual increase of feebleness, no desire to eat or take exercise; children have no desire to play or study. Early symptoms are vomiting and insomnia or disturbed sleep. There may also be indigestion. The temperature is either normal or slightly elevated. There are involuntary movements of sucking or mastication, the reflexes are exaggerated, and the muscle tonus is generally increased. The pulse is slow, the pupils are narrow or uneven, reaction to light may be maintained, incomplete, or entirely absent.

There is at first light coma, which, in the further course of the disease, increases to deep unconsciousness. The patient is unable to swallow; there is motor irritation in the shape of involuntary muscular twitching of the face, trunk, and extremities, which terminates in paralysis, increasing debility, and death.

Diagnosis.—The diagnosis of tuberculous otogenic meningitis requires considerable experience, especially in the early stages, as the symptom-complex is often incompletely developed or entirely absent in the latent period. Middle-ear suppuration setting in and progressing without any symptoms points to its tuberculous character. Another characteristic point is the presence of a particularly small perforation, with copious and very fetid suppuration which has persisted for a number of years. A tuberculous family taint will often arouse our first suspicion of tuberculous meningitis. The exact diagnosis can be made by lumbar puncture, the exudate showing a network of minute coagula-

tions (Fig. 117). Careful examination will also demonstrate the tubercle bacillus in 75 per cent. of the cases.

Treatment.—An ear operation offers no chance of success. Temporary improvement is sometimes achieved by lumbar puncture, daily repetition of which has been recommended. Withdrawal of blood from the mastoid by the application of leeches, inunction with green soap, and creosote internally, have also been recommended.

Course.—Tuberculous meningitis lasts from one or two weeks to several months.

In cases which run a protracted course, temporary improvement is not a rare occurrence. The fully developed symptoms may be preceded by a long state of irritation resembling serous meningitis. The latter is apparently cured, but, after a subjective feeling of well-being for several weeks or months, the signs of tuberculous meningitis set in. Aside from exceptional cases of improvement which have been reported, the course of the disease is always fatal.

VII. OTOGENIC ABSCESS OF THE TEMPOROSPHENOID LOBE

Anatomy.—The abscess is usually located in the basal part of the temporosphenoid lobe, more or less turned toward the tegmen tympani. Toward the outside it extends to the cortex of the temporal lobe, reaching the medial and sometimes the upper temporal convolution (Fig. 113). Medially the abscess extends in the direction of the descending horn of the lateral ventricle and the crus cerebri; anteriorly it extends to the operculum (Plate IX); posteriorly it varies considerably in extent. In most cases the abscess does not project beyond the tympanosphenoidal lobe, and it is only in exceptional cases that it continues to the anterior portion of the occipital lobe.

The size of these abscesses is from a hemp-seed, in suppurative superficial encephalitis, to a man's fist. They are spherical or oval, rarely irregular or ramified. Transition forms of multiple abscesses, which intercommunicate by small canals, are rare.

The pus in a cerebral abscess is under considerable pressure, and usually completely fills the abscess cavity. It is mostly putrid and rarely of liquid consistency.

There are always necrotic cerebral and fibrinous substances mixed with it, all of which are evacuated together. The exuding pus is in most cases fetid, non-fetid pus being only occasionally found in acute and such chronic cases as have been opened in the latent stage. In the manifest stage which very often occurs by acute endocranial reinfection the pus is generally ichorous.

The pyogenic factors vary considerably in different cases. Usually there is but one species of micro-organism present in any one case,

together with anaërobic bacilli. Cerebral abscesses with mixed infections, or containing the colon or proteus bacillus, occur less often. A study of a large number of cases, however, will show that nearly all kinds of pyogenic factors may occur in abscesses of the temporal lobe. In chronic cerebral abscesses the culture may remain sterile, which is undoubtedly due to the destruction of the micro-organisms previous to evacuation, so that they can no longer be demonstrated.

Abscesses of the temporal lobe are usually enclosed in a fibrinous capsule which fairly well protects the neighboring cerebral substances from infection. The capsule is smooth toward the contents of the abscess, there being no protrusions. In cases which have persisted for

Fig. 113.

Abscess of temporo-sphenoidal lobe; chronic suppurative pachyleptomeningitis after intradural perforation of abscess (*a*).

some time the capsule contains an abundance of pigment. The independence of the capsule is in many cases demonstrated at operation or during the healing process. It is usually spontaneously expelled a few days after the abscess has been emptied.

The cerebral substance in the vicinity of the abscess is in all cases more or less compressed. There may be displacement and change of form of the lateral ventricle, even compression of the crus cerebri in the region of the lemniscus or at the emergence of the trochlear nerve. The cerebral substance in the vicinity of the abscess contains small round-cell infiltration, and there are various stages of cell necrosis and thrombosis of the smallest cerebral vessels. Characteristic cross sections of an otogenic abscess of the left temporal lobe are illustrated on

Plate IX. The size of the abscesses is in some cases more dependent on displacement of the neighboring tissue than on suppurative degeneration of the cerebral substance. This fact is demonstrated by anatomical examinations of the bone in cases of abscess of the temporal lobe, showing, as they do, that in the region of the abscess but a small part of the fibres has been completely destroyed, the larger portion being displaced or compressed in the neighborhood of the abscess. This anatomical fact is also confirmed by clinical experience, operative removal of an extensive abscess of the temporal lobe being always followed by such a rapid reduction of the abscess cavity that in from one to two weeks after operation a cavity the size of a small apple may have been reduced to that of a prune. This reduction can, of course, not be the consequence of tissue regeneration in so short a time, and, besides, local examination will show that it is but to a small extent produced by blood coagula or fibrinous masses. The fact is that by evacuation of the abscess the pressure on the brain has been removed and the displaced parts are returning to their normal location.

After the abscess has persisted for several weeks there will be softening and œdema of the cerebral substance in the more or less distant vicinity of the abscess, which is evidently occasioned by congestion. The brain assumes a pathologic reddish tint; the cerebral cortex becomes softened and is easily compressible.

The soft parts between the temporal bone and the basal surface of the abscess show various changes according to the origin of the abscess. Direct spreading of the suppuration from the epitympanum to the brain is only rarely responsible for the formation of the abscess. Where it does occur, the process is preceded by pachymeningitis externa and interna and by superficial suppurative encephalitis. All these cases of cerebral abscess are complicated by an intradural abscess, and most of them by an extradural abscess. The lymph-spaces between the pachymeninges and the cerebral surface are replete with pus, or in older cases are obliterated by connective tissue, owing to the secretion of fibrinous masses. A fistulous canal, filled with pus, leads from the auricular region into the cerebral cavity.

In cerebral abscesses of metastatic origin the soft layer between the base of the abscess and the bones (labyrinth, tegmen) is at first unchanged except for the œdematous swelling of the brain. Later there will be congestive manifestations of the dura; the dural vessels are full to turgescence, finally leading to softening, swelling, and inflammation of the dura. A fibrinous communication between the inner surface of the dura and the arachnoidea will establish itself, causing the formation of a thick, callous layer between the temporal bone and the lower surface of the abscess, in the event of the latter running a chronic course.

Anatomical Course and Result.—There is a theoretical possibility for a spontaneous cure of cerebral abscess taking place by calcareous degeneration of the purulent contents and obliteration by connective tissue, but I have never been able to observe such a case. Once the abscess has grown to the topographical limit and its inside pressure is sufficiently high, the usual result is perforation into the neighboring parts. Such perforation may even occur in relatively small abscesses when located near the cerebral surface of the lateral ventricle. The perforation occurs outward into the intradural or subdural space or inward into the lateral ventricle. The former event will cause diffuse infectious suppurative meningitis, as long as the normal communication of all subdural spaces still exists, or, should the region of the perforation be closed against the other subdural spaces by fibrinous or connective-tissue adhesions, an intradural abscess will develop. In the presence of a fistula, however, leading outward or into the middle ear, the cerebral abscess may evacuate outward and lead to a cure. This favorable result may sometimes be observed clinically on the occasion of a radical operation in the stage of latency and simultaneous exposure of the dura. Thus, I observed spontaneous evacuation of a cerebral abscess from a rupture which occurred when changing bandages, without giving rise any symptoms.

Perforation of an abscess of the temporal lobe into the lateral ventricle always causes the development of infectious suppurative meningitis.

The behavior of the cerebral meninx and of the cerebrospinal fluid in the presence of an abscess of the temporal lobe deserves special consideration. It has already been stated that there will be early œdema of the brain and inflammatory softening as well as thickening of both the dura and pia, especially in the region of the abscess base. These meningitic changes, which occur as concomitant symptoms of the abscess formation, are accompanied by typical changes in the cerebrospinal fluid. Lumbar puncture in cerebral abscess yields a fluid under pathologically increased pressure, even if the abscess is not very large and closed in on all sides. The fluid is turbid and discolored gray or grayish yellow. After six to twenty-four hours it will form minute fibrinous coagulations in the undisturbed test-glass, and the microscopic examination of the sediment—obtained with or without the centrifuge—shows an abundance of mono- and polynuclear leucocytes. Micro-organisms, however, are not present, either microscopically or by culture, unless there is a perforated cerebral abscess. The lumbar fluid, therefore, is sterile. This suppurative, non-infectious form of meningitis, which may also be present in other intracranial otogenic affections, is a typical and very valuable symptom for the differential diagnosis of most otogenic abscesses of the temporal lobe.

The reduction of the abscess cavity after evacuation of its contents is occasioned by the extension of the neighboring cerebral parts which had been compressed by the abscess, and by granulations which gradually become organized. The remaining gap will be gradually closed by connective tissue which starts growing from the dura, but the number of cases where the former abscess cavity has been completely obliterated is very small. In most cases small cysts will persist in the region of the incision or perforation, which are filled with a clear liquid containing normal or degenerated round cells. It may be possible that an acute middle-ear suppuration will infect these cysts, causing fresh abscess formation or suppurative meningitis. This is a contingency that should be duly considered in treating the wound and making the prognosis.

Symptoms.—The symptoms to be expected on the part of the affected auditory canal are but few and in no case very characteristic or prominent. Nor are there any particular changes to call the patient's attention to the gravity of the complication, unless by way of exception. The origin of an otitic abscess of the temporal lobe is a suppurative infection of the upper space of the tympanic cavity in most cases, thus representing the epitympanic type of a middle-ear suppuration which is nearly always chronic. Inflammatory mastoid manifestations do not belong to the typical picture of a suppuration of the middle ear complicated by an abscess of the temporal lobe.

Against these typical cases there are a small number of others in which the abscess of the temporal lobe occurs as the result of a chronic middle-ear suppuration with copious fetid secretion, involving all the middle-ear spaces.

The pathological process as such commences almost without exception in an insidious way under vague general manifestations (feebleness, lassitude, anorexia, diffuse headache, sometimes nausea and vomiting). The picture may at first be complicated by ear symptoms. If the latter have led to suppurative degeneration of the tegmen tympani, even transitory retention of pus will cause headache, a sensation of heaviness in the head and of fulness in the ear, which may be accompanied by violent subjective noises. In such cases, therefore, where the abscess has developed through direct spreading of the ear suppuration, these symptoms are the precursors of the abscess symptoms, from which it is sometimes impossible to differentiate them. In an abscess of the temporal lobe which has developed by metastasis there may be no auricular symptoms whatever.

There is nothing pathologically characteristic in the body temperature, which is either moderately raised or subfebrile, in rare cases normal, or in the latent stage sometimes subnormal. In a few cases there is fever of a pronounced intermittent character. Perforation of the ab-

scess is nearly always associated with a considerable rise in temperature, up to 104° or more, sometimes complicated by chills.

In the further course of the disease there will be symptoms to be attributed to increased cerebral pressure or to cerebral changes in the direct vicinity of the abscess (cerebral œdema, encephalitis). They consist in headache, irregular and retarded pulse in the initial stage, cranial susceptibility to percussion, congestive manifestations of the ocular fundus, nausea, vomiting, narcolepsy, disturbed consciousness, delirium, tonic and clonic spasms.

In the acute stage of the abscess there are pathological symptoms which are caused by the destruction of cerebral substance and by interruption of the nerve conduction (focal symptoms). These include spastic and paralytic manifestations of the facial muscles of the same side and of the extremities on the opposite side. Simultaneous manifestations on the part of the facial muscles and extremities occur in pathological foci located in the internal capsule. In right-handed individuals abscesses of the left temporal lobe may lead to lingual disturbances (amnestic aphasia, paraphasia, conduction aphasia, and in rare cases to optic aphasia and word-deafness). Amnestic and motor agraphia is not an unusual occurrence. There may also be paralysis of the oculomotorius, as evidenced by ptosis, mydriasis, and divergent strabismus. Pressure of the cerebral abscess upon the crus cerebri causes early disturbances in the region of the trochlear nerve on the affected side. Simultaneous paralysis of that nerve may be regarded as a direct characteristic sign for an otogenic abscess of the temporal lobe. There may also be irritative and paralytic manifestations on the part of other cerebral nerves, notably disturbances of smell (anosmia). On the other hand, symptoms of those cerebral nerves which leave the skull through the posterior cranial fossa are usually absent. Symptoms in the region of those nerves and rigidity of the neck will not occur unless diffuse suppurative infectious meningitis has developed from perforation into the ventricles or subdural spaces. In unilateral auricular affections the hearing acuity of the other, normal, ear may be impaired, owing to a lesion of the crossed hearing centre in the affected temporal lobe.

The complete course of symptoms corresponds to a division into four clinical stages: (1) initial stage, (2) latent stage, (3) manifest stage, (4) terminal stage.

The initial stage is characterized by general symptoms such as anorexia, lassitude, fatigue, dislike to work, and sometimes vomiting after ingestion of food. The picture may also be complicated by auricular symptoms. Furthermore, there are usually diffuse headache in the neighborhood of the affected ear, vertigo, disturbed consciousness at

night lasting for a few minutes, short slight spasms of the extremities of the opposite side, transitory fever and delirium. The pathological symptoms in the initial stage, lasting for two or three weeks, will gradually disappear.

In the latent stage, which lasts from three to twelve weeks, the patient may feel tolerably well, but usually disturbed sleep and a feeling of infirmity will persist. The patient looks depressed and is easily fatigued after short, slight muscular exertion.

The latent stage nearly always passes into the acute stage without any warning. For instance, after a more than usually restless night a grave pathological picture may suddenly be present, consisting in vomiting, delirium, spasms of the extremities, and twitching of the facial muscles. There are lingual disturbance, paralytic and other disturbances of vision, and increase of the spasms, vomiting, and delirium. This stage is usually accompanied by tormenting, localized headache, considerable susceptibility of the skull to percussion, fever, delirium, exaggerated reflexes, general hyperæsthesia, and ankle clonus. The acute stage, which lasts from one to fourteen days, may pass into the terminal stage under almost imperceptible exacerbations or quite suddenly.

The symptoms of the cerebral abscess are now less pronounced than those of otitis. Coma takes the place of delirium, spastic paroxysms no longer occur, but there is paralysis of the extremities of the affected side, paralysis of the oculomotorius, exophthalmos, frequent cerebral vomiting, diarrhœa, and incontinence of fæces and urine. Death occurs in deep coma after paralysis of all motor and sensory functions has further increased.

Diagnosis.—The diagnosis of abscess of the temporosphenoidal lobe is difficult in the first stage, but a careful examination of the ear will always have to be made if a perfectly satisfactory explanation of the general and local complaints cannot be detected by an internal examination of the body. An experienced observer will then be able in many cases to diagnosticate an intracranial complication. An exact differentiation from circumscribed meningitis and an extradural abscess, however, will not be possible in this stage, nor in any subsequent stage. From a practical point of view, however, such a differential diagnosis is immaterial. The important diagnostic point is to establish intracranial complication in the area of the middle cranial fossa, from which the necessity of immediate surgical intervention would clearly follow. The experienced otological surgeon will then be able to make an exact diagnosis at the operation, from the dural changes and the exposed brain, and, according to the findings, proceed to the opening of the abscess.

There can be no question of a clinical diagnosis of abscess of the temporal lobe during the latent stage. Experience, however, shows

that it is just during this stage that many abscesses can be successfully operated upon. The discovery of the abscess is nearly always accidental on the occasion of a radical operation which may become necessary from some other pathological symptom.

The following case may serve as an example:

In a case of chronic suppuration of the attic, conservative treatment is persisted in for a long time; the fetid character of the suppuration is not improved and there is headache. Radical operation is now decided upon, and the roof of the tympanic cavity is found softened from suppuration, while the middle-ear spaces contain but very little pus. These findings would not explain the grave complaints. Upon resection of the middle cranial fossa, a considerable vascular injection suggests the proximity of the abscess, which is now uncovered and evacuated by incision.

I have also operated upon two cases where after the radical operation the abscess spontaneously evacuated through a pathological fistula during the process of healing. In one of the cases the radical operation was carried out upon the relative indication of persistent fetid suppuration of the middle ear, the bone being likewise demonstrably involved. Hearing acuity of the other ear was normal. The operation disclosed osseous changes of the roof of the tympanic cavity, which was therefore removed. The healing process took a perfectly normal course; the patient felt perfectly well; but during a change of bandage there was spontaneous perforation and evacuation of the cerebral abscess outward. The other case was similar. After operation the patient stated that he suddenly felt large quantities of fluid flowing into his ear. Immediate change of bandage showed that a latent abscess of the temporal lobe had been evacuated into the bandage. In both cases the abscesses were large, containing about 80 c.c. of pus.

The diagnosis in the acute stage usually presents no difficulties. The choked disk and the paralysis of the trochlearis, which can be recognized by careful examination, are valuable symptoms. To these are added the characteristic lingual disturbances in right-handed people with abscess on the left side. Besides, the other symptoms mentioned above are not present in such characteristic relationship in any other endocranial affection as in abscess of the temporal lobe.

The clinical diagnosis of cerebral abscess is often impossible in the terminal stage, since all that can be recognized is the purulent meningitis it has caused. The only way to discover these abscesses is to resort to operation in purulent otitic meningitis. The idea of regarding purulent meningitis as inoperable may at the present time be looked upon as absolutely exploded: it was owing to this very idea that many a case of abscess of the temporal lobe remained unoperated upon in the ter-

minal stage, because of the erroneous diagnosis of suppurative diffuse meningitis. Thus, the presence of a cerebral abscess, opening of which might have saved the patient's life, was only discovered at autopsy.

Differential Diagnosis.—In the initial stage all the conditions should be considered which are accompanied by general, though not pronounced, symptoms, such as dejection, fever, headache, fatigue on slight exertion. Very often a diagnosis is made of "cold," indigestion, hysteria (!), anæmia, etc., and it is in this stage that mistaken diagnoses are of such frequent occurrence that the number of correct ones are decidedly in the minority. An early diagnosis of an intracranial affection is often possible by examining the ocular fundus, which should be done in all cases.

A differential diagnosis between abscess of the temporal lobe and any other otitic affection of the medial cranial fossa before operation is often impossible in the terminal stage, although lumbar puncture may be of assistance. This requires not only microscopic examination, but also observations as to coagulation and culture experiments. This, however, cannot be accomplished in less than one to two days, and, as the operation is exceedingly urgent, it does not seem advisable to defer it for that length of time.

Treatment.—The treatment of otogenic abscess of the temporal lobe is surgical. It consists in opening the middle cranial fossa, exposure and opening of the regional dura, and evacuation of the abscess. As a preliminary act, antrotomy is done in cases of acute suppuration of the middle ear, and the radical operation in chronic cases.

If the general condition of the patient is unfavorable, these operations must be carried out as rapidly as possible. I am in the habit in such cases of deferring the plastic of the auditory canal until a later time, in order to get as rapidly as possible to the middle cranial fossa.

Exposure of the dura may be effected in the following ways:

(1) Starting from the squama of the temporal bone.

(2) Removal of the roof of the tympanic cavity.

(3) Removal of the tympanic roof, the upper wall of the auditory canal, and the neighboring part of the squama.

The first method is the oldest. In this now discarded way the abscess can only be discovered in very few cases, because the bone is exposed at a point far away from the otogenic origin of the abscess. In former times it was even customary to confine the operation to this insufficient measure, omitting the operation on the ear entirely. The consequence is that the cause of the abscess will persist. Even should it be possible to evacuate the latter, the suppuration of the ear will continue, leading to postoperative, rapidly fatal meningitis; otherwise, the patients will succumb to a recurrence of the cerebral abscess.

The second method, which consists in gaining access to the middle cranial fossa by removing the tegmen tympani, unquestionably offers the best possibility of finding the abscess and of establishing sufficient drainage of the abscess cavity, as it follows the track over which the infection has occurred. The method, however, has many disadvantages. The deep horizontal aperture does not admit of freely surveying the cerebral changes themselves. In metastatic formation of the abscess it will be necessary to explore the brain with the scalpel in various directions. The opening of the tegmen, however, considerably interferes with exploration toward the posterior part of the temporal lobe and the occipital lobe; in fact, exploration in certain directions is quite impossible. It is not surprising, therefore, that in metastatic abscesses of the temporal lobe removal of the tympanic roof will not attain the desired end.

Under these circumstances clinical requirements have led to the development of the third operative method, which we now employ in every case. We commence with the exposure of the middle cranial fossa by removing the roof of the tympanic cavity, the upper wall of the auditory canal, and the neighboring part of the squama. From the aperture thus obtained the bone is resected laterally to the zygomatic process and to the base of the squama. This admits of a full survey of the region and of inspecting the affected cerebral cortex.

By carefully lifting the dura it can be ascertained whether there is an extradural abscess or not. If so, the dura is opened at the most affected place, either by a longitudinal incision of 1–2 cm. or by a crosswise incision. Larger dural vessels should be avoided, in order to prevent the occurrence of disturbing hemorrhages. The tension of the dura varies. If the abscess has not yet perforated, the dura is hard, elastic, and very tense. The tension is increased considerably beyond normal. If perforation has taken place, there may be distinct fluctuation with high pressure; if it has occurred into the subdural spaces, the dura may be quite relaxed, with much fluctuation, and may even be synchronous with the pulse.

Incision.—The dura is incised layer by layer, until the bone is exposed; the latter is then also incised. By following this rule it will be possible to distinguish from which region the pus emanates and whether the abscess had or had not subdurally perforated. The pus, intermingled with brain particles, rapidly rushes out, or even spurts out if the tension is very high. A little later it exudes with the pulse movements. Evacuation of the abscess is usually followed by lumpy, fibrinous masses. The cavity should neither be washed nor dried; it is simply drained by means of iodoform or isoform wicks.

The first change of bandage is made two to three days after the operation. The drains are shortened on this and every subsequent

PLATE IX.

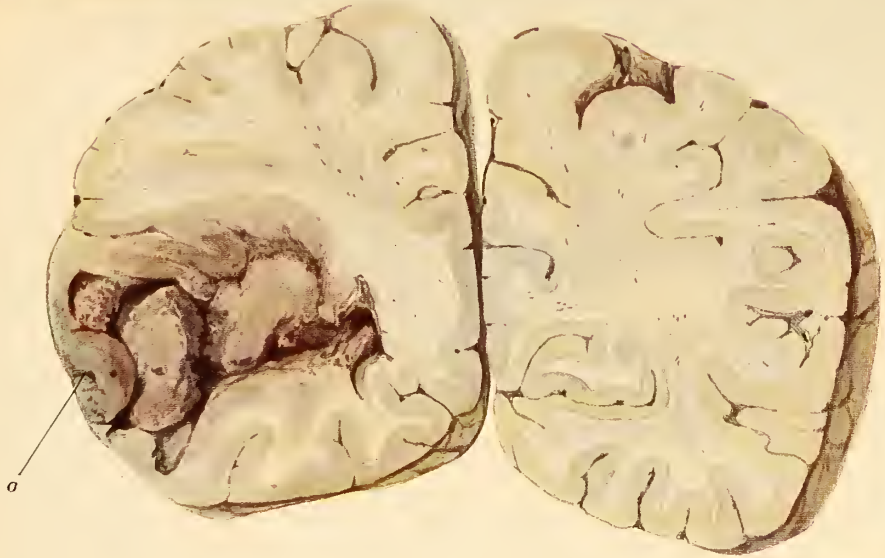


FIG. 1.
The abscess is in the anterior portion.

FIG. 2.

FIG. 3.
d Topography of otogenic abscess of the tympano-sphenoidal lobe.

Anterior vertical sections through the anterior part (Fig. 1), the middle part (Fig. 2), and the posterior part (Fig. 3) of an abscess of the left hemisphere. Formalin preparation. *a*, intact cerebral cortex; *b*, flat roundish œdematous swelling of the temporal lobe in the medial abscess region; *c*, perforation; *d*, extension of abscess toward the descending horn and the inner capsule.

occasion, so that the drains will not be entirely removed until eight to ten days after operation. Further treatment should take the rapid healing of the dural aperture above the tympanic roof into consideration. With this end in view, the abscess cavity is drained through the upper outer end of the aperture in the area of the temporal squama. It will then be possible to undertake the undisturbed treatment of the trauma caused by antrotomy or the radical operation, quite independently of the cerebral opening.

Treatment of the abscess cavity from the tympanic cavity would, of course, cause the dural aperture above the tympanic cavity to persist for a long time, with the consequence of an atropic scar of the dura. Besides, the dural trauma is always covered over by cicatricial tissue which develops during the healing process of the middle ear and which, in the presence of an atropic dura, may lead to headache requiring a plastic operation for removal. On the other hand, the dura has already healed with scar-formation in the area of the tympanic roof and antrum if the abscess cavity is treated from without.

Patients generally make a rapid recovery, their appearance is excellent, and they rapidly increase in weight; but for a long time afterward they are entirely unfit for any intellectual or physical work.

The prognosis is not unfavorable; it chiefly depends upon the age of the patient and upon whether the abscess has already undergone perforation or is still entirely closed. In older individuals the prognosis is less favorable than in young ones. Cases in which the abscess is still entirely closed give better chances for an operation than where perforation has taken place. Perforation of the abscess toward the ventricle renders the prognosis absolutely unfavorable. With perforation into the subdural spaces, healing may still take place, even in the presence of suppurative meningitis.

VIII. OTOGENIC CEREBELLAR ABSCESSES

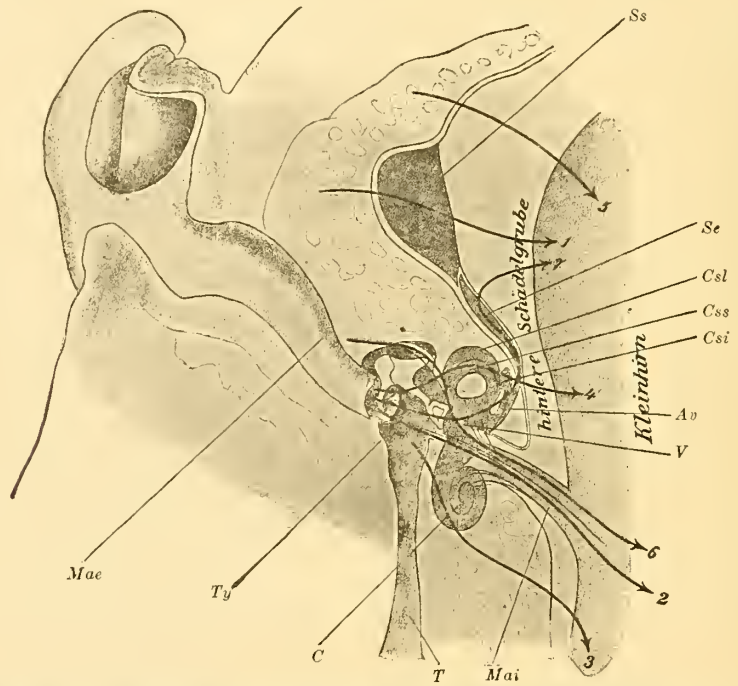
Anatomy, Etiology, and Occurrence.—Otitic cerebellar abscesses are rarely observed in the course of acute middle-ear inflammation. It always occurs in complicated acute middle-ear suppuration which has already led to suppurative mastoiditis, extradural abscess, intradural abscess, or pyæmic sinus thrombosis. Exceptionally a tuberculous cerebellar abscess may develop through ulceration or cerebellar tubercles in cases of subacute tuberculous middle-ear suppuration.

In chronic middle-ear suppuration a cerebellar abscess develops in most cases on the basis of chronic or acute diffuse suppuration of the labyrinth.

Fig. 114 illustrates the infectious tracts which may play a part in the spreading of middle-ear suppuration to the posterior cranial fossa.

It will be seen that 5 of the 7 tracts—namely, Nos. 2, 3, 4, 6, and 7—pass through the labyrinth, and in these cases the cerebellar infection is preceded by suppurative inflammation of the labyrinth, whether the abscess has developed by spreading of the suppuration or metastasis.

Fig. 114.



The infectious tracts leading from the ear into the posterior cranial fossa. (Horizontal section through the ear and the posterior cranial fossa.) *Ss*, sinus sigmoideus; *Se*, saccus endolymphaticus; *Csl*, canalis semicircularis lateralis; *Css*, canalis semicircularis superior; *Csi*, canalis semicircularis inferior; *Av*, vestibular aqueduct; *V*, vestibulum; *Mai*, internal auditory meatus; *T*, eustachian tube; *C*, cochlea; *Ty*, tympanum; *Mae*, external auditory meatus.

The various infectious tracts are denoted by figures.

(1) The suppurative inflammation of the mastoid continues to the sinus sigmoideus, whence it penetrates into the cerebellum.

(2) The inflammation extends from the middle-ear spaces (*Ty*) through the cochlear window, vestibulum, and cochlea, to the internal auditory canal and along the nerve tuft of the internal auditory canal into the cerebellum.

(3) Suppurative otitis media with fistula of the promontory. The complication of the posterior cranial fossa occurs through an endocranial fistula of the cochlea.

(4) Conveyance of the suppuration from the middle ear to the posterior cranial fossa through an endocranial fistula of the upper or posterior semicircular canal, which are likewise infected. The area of the middle cranial fossa may become involved by the formation of an endocranial fistula of the vertex or anterior part of the superior semicircular canal.

(5) Extension of mastoid suppuration to the posterior mastoid cells in the sinus region, followed by perforation into the posterior cranial fossa.

(6) Spreading of suppurative otitis media to the posterior cranial fossa through the canalis facialis.

(7) Otitis media leading to ulceration of the labyrinth either direct or through a fistula. The spreading of the labyrinth suppuration to the posterior cranial fossa takes place through the vestibulum, the vestibular aqueduct, and the ductus and saccus endolymphaticus.

Cases with chronic suppurative ostitis of the temporal bone and those complicated by cholesteatoma are particularly dangerous in causing cerebellar abscess, as soon as they have led to a suppurative inflammatory involvement of the labyrinth and to acute suppurative disintegration of the cholesteatoma.

Some cerebellar abscesses are enclosed in a fibrinous capsule, but most of them are not. Those without a capsule are often of irregular shape (Fig. 116) and are provided with blind rami and sinuses.

After an abscess of this kind has persisted for some time, it is not unusual for several daughter abscesses to develop in the same hemisphere, in the worm, or exceptionally in the opposite hemisphere. Œdema and serous osmosis from the adjacent meninges occur as early changes in the immediate vicinity of the abscess. Later, fibrinous secretion will occur in the intradural space, adhesion of the meninges, or in rare cases extensive callosities. The anatomical result of a cerebellar abscess which has been left alone consists in outward perforation into the intradural space (Fig. 115) or into the fourth ventricle. In both cases the immediate consequence is fatal suppurative meningitis.

Fig. 115.

Girl, thirteen years old. Abscess of the right cerebellar hemisphere (a) near the perforation, with regional swelling of the right hemisphere and discoloration of the surface.

Cerebellar abscess occurs more frequently in the second decade of life than in any other.

Symptoms.—All cases occur in conjunction with an otherwise complicated fetid middle-ear suppuration; in chronic cases there is usually acute suppurative decomposition of a cholesteatoma. Inflammatory mastoid manifestations are usually present in acute cases, while in chronic cases the mastoid may be perfectly normal. Pus retention in the antrum is of frequent occurrence, leading to lowering of the posterior wall of the osseous auditory canal or fistulous perforation. In the simultaneous presence of labyrinth suppuration, which occurs in about 85 per cent. of all cerebellar abscesses in the course of chronic middle-ear suppuration, all the symptoms of diffuse labyrinth suppuration (vomiting, vertigo, equilibrial disturbances, deafness, spontaneous nystagmus, fistular symptoms, etc.) can be established by examination or history.

There is nothing characteristic in the otoscopic findings, which rather show the various forms of the types found in complicated suppuration of the middle ear.

General Symptoms.—There are physical and psychic unrest, fatigue, insomnia in spite of a great desire to sleep, severe occipital headache, frequent vomiting of the cerebral type, and paroxysms of vertigo.

Further early symptoms are susceptibility to pressure at the nape, more or less rigidity of the neck and susceptibility to percussion at the occiput.

FIG. 116.

Horizontal section through the cerebellum and the crus cerebri at the level of the corpora quadrigemina. Irregular, old abscess (*a*, *a'*) in the right cerebellar hemisphere. Its anterior portion (*a*) extends to the surface of the hemisphere.

Girl, twelve years old (chronic suppuration of the middle ear and labyrinth, acute exacerbation by streptococcus pyogenes). Death from acute suppurative meningitis.

In advanced cases there are general debility, decrease in muscular power, depression, anorexia, sallow complexion, and flabbiness of the skin. Fetid middle-ear suppuration which has existed for a long time subjects the patient to malnutrition, causing an anæmic appearance.

As to cerebellar and endocranial symptoms, cerebellar ataxia should be mentioned in the first place. It is characterized by disturbances of coördination, especially of the trunk and the lower (sometimes also of the upper) extremities of the affected side (hemiataxia), which can be recognized while standing, or walking backward and forward. They are, however, often concealed by equilibrial disturbances due to the labyrinth. Cerebellar ataxia is more distinctly demonstrated on examining the coördination of the extremities in lateral walking.

Cerebellar nystagmus is very slow and coarse, in most cases straight-lined and horizontal. It is generally directed toward the affected side; the direction is changeable and nearly always attains a high degree of intensity.

A cerebellar symptom of great importance consists in abducens paresis or paralysis of the affected side (caused by cerebral œdema or pressure of the abscess on the abducens nerve) and double vision. Disturbances in the region of the oculomotor nerve and paralysis of vision are less frequent.

In cases which have existed for a long time there are usually opisthotonos and stupor. The ocular fundus is nearly always changed, usually more or exclusively on the affected side. The veins are plethoric, but a pronounced choked disk occurs only after the abscess has existed for a long time and especially when it is complicated by suppurative meningitis. The cerebrospinal fluid is usually turbid and contains microscopic pus corpuscles. In culture it is generally sterile, pathogenic micro-organisms being found only in a small percentage of cases. Spasms of the muscles of the face, neck, and extremities are always indications of the cerebellar abscess having led to diffuse meningitis.

The temperature of the body is either normal or subnormal, but suddenly rises considerably in the last stages of fatal cases. The pulse is retarded, respiration accelerated and often irregular. In advanced cases there are sometimes bulbar disturbances of speech and Cheyne-Stokes respiration. There is usually albuminuria in the final stages, and sometimes sugar in the urine.

Course.—The course of a cerebellar abscess may be divided into four stages, like that of the temporal abscess. The initial stage is characterized by the general symptoms explained above. The predominant focal symptoms are obscured by the labyrinth symptoms in the presence of suppuration of the labyrinth. Homolateral cerebellar ataxia, if present, is a valuable symptom. The initial stage will last from one or two weeks to one or two months. In the latent stage the subjective feeling of the patient is good, and it requires a careful examination to demonstrate the existence of unilateral ataxia and disturbances of coördination of the extremities of the affected side. This stage lasts from a few days to a few weeks. The transition of the latent into the acute stage is usually shown by the occurrence of double vision or abducens paralysis. There are also all the above focal and general symptoms. This stage lasts from a few days to about two weeks. In the terminal stage the symptoms of perforated abscess or purulent meningitis prevail.

Diagnosis.—The clinical diagnosis of cerebral abscess meets with considerable difficulties in all stages.

Careful testing for cerebellar ataxia, lateral walking toward the affected side, abducens paralysis with double vision, may offer valuable

guiding points. In the latent and terminal stages an exact clinical diagnosis is out of the question. But it is possible in nearly all cases to establish the presence of an affection of the posterior cranial fossa. An experienced surgeon will then be led, by the operative findings of the labyrinth, dura, or sinus sigmoideus, to proceed to the cerebellum and evacuate the abscess.

Differential Diagnosis.—The following affections come in for consideration: (1) suppuration of the labyrinth; (2) circumscribed meningitis of the posterior cranial fossa; (3) extra- and intradural abscesses of the posterior cranial fossa; (4) suboccipital suppurations, which are usually of a tuberculous nature; (5) tumor of the auditory nerve; (6) cerebellar tumor.

A cerebellar abscess is sufficiently differentiated from suppuration of the labyrinth by the presence of hemiataxia, lateral walk to the affected side, and the unilateral disturbances of coördination of the extremities. The differential diagnosis will be more difficult in a patient who is bedridden owing to frequent severe paroxysms of vertigo, and cannot be tested for standing or walking. The question, however, is not so much as to whether there is any cerebellar abscess or a suppuration of the labyrinth, but rather whether there is any suppuration of the latter alone or associated with a cerebellar abscess. If the symptoms of the former have already disappeared, the differentiation will be easy. If vertigo due to the labyrinth has already been removed by operation, the persistence of vertigo would point to the presence of a cerebellar abscess.

Furthermore, spontaneous nystagmus is always directed toward the non-affected side in uncomplicated diffuse suppuration of the labyrinth, while the reverse is the case in cerebellar abscess.

Cerebellar nystagmus is horizontal and very intense, while that occurring in diffuse suppuration of the labyrinth is coarse or fine, often rotatory, and, in chronic cases, of the lowest or medium degree of intensity.

Differentiation between cerebellar abscess and circumscribed meningitis of the posterior cranial fossa or intradural (intrameningeal) abscess depends upon the history. Circumscribed meningitis of the posterior cranial fossa has no latent stage, so that the symptoms undergo continuous exacerbation. In cerebellar abscess, however, there are periods of relief and often transitory disappearance of the symptom-complex. It should be specially mentioned, however, that cerebellar abscesses are often associated with extra- or intradural abscesses of the posterior cranial fossa.

Suboccipital suppuration starts from osseous foci of the base of the skull (occipital bone or petrous bone) or from the first two cervical vertebræ. There is usually complete rigidity of the neck, the patient being

unable to raise or turn his head except with the assistance of his hand. The differential diagnosis is still further facilitated if there is no suppuration of the ear, whether simple or complicated. As was explained above, a cerebellar abscess presupposes a further complication of a middle-ear suppuration, notably by a suppuration of the labyrinth or a purulent affection of the dura in the region of the cerebellar hemisphere (dura, venous sinus). X-ray examination will likewise furnish exact information.

As to a tumor of the auditory nerve, the differential diagnosis is only of clinical importance if in the course of growth it has led to a compression of the cerebellar hemisphere or to cerebellar symptoms. The most important sign is the absence of a suppurative inflammation of the ear in the presence of a tumor as against the presence of a chronic suppuration of the middle ear or labyrinth in cases of cerebellar abscesses. Besides, large tumors of the auditory nerve lead to early compression of the pons, spasms of the extremities, paresis, conjugate paralysis of vision, and choked disk of one or both eyes. The functional test of the internal ear in the presence of an auditory tumor reveals nearly always complete deafness and unexcitability of the static labyrinth. There are the same findings in diffuse suppuration of the labyrinth which may have led to a cerebellar abscess. Consequently, the functional test of the internal ear will only aid the differential diagnosis if there are still positive hearing ability and reflex excitability. Such findings are against the assumption of auditory nerve tumor. The presence of peripheral paresis of the facial points rather to suppuration of the internal auditory canal than to tumor. A facial nerve which is completely embedded in tumor masses, or is even adherent to the tumor, often remains capable of function.

The differential diagnosis between cerebellar tumor (sarcoma, glioma, gumma) and cerebellar abscess is impossible if the tumor has the topographic location of the abscess and there is a suppurative affection of the ear. The differentiation between cerebellar tubercles and an abscess is equally impossible under similar circumstances. In most cases, however, a cerebellar tumor leads to bilateral cerebellar ataxia, bilateral abducens paralysis, and choked disk, while the etiologically important middle-ear suppuration is absent.

Treatment.—This consists in the operative exposure of the posterior cranial fossa, division of the dura, opening of the cerebellar hemisphere, and evacuation of the abscess. It is advisable in operating to follow the track of the suppuration. It is necessary, therefore, in all cases of labyrinthogenic cerebellar abscess that the radical operation be followed by resection of the ulcerated labyrinth previous to opening the posterior cranial fossa.

Cerebellar abscesses which have developed from a deep extradural abscess of the posterior cranial fossa or from thrombophlebitis of the

sinus sigmoideus are best opened from the region of the sinus and from the part of the dura between the sinus and labyrinth, provided the latter is intact.

The incision of the posterior cranial fossa should be as deep as possible, so as to prevent retention of pus at the base of the abscess cavity or at the fundus of the cranial fossa, and to enable the operator to open and drain the abscess at its lowest point. The abscess cavity is then loosely packed with iodoform wick. The first change of bandage should take place on the second day after operation.

Postoperative Prognosis and Course.—The operative success in otitic cerebellar abscesses is by no means satisfactory as yet, there still being a mortality of about 40 per cent. It is only exceeded by otitic meningitis among all other otitic endocranial affections. The prognosis is much less favorable than that of abscess of the temporal lobe or pyæmic sinus thrombosis. This is explained by the fact that in nearly all cases a cerebellar abscess is not the only endocranial otogenic affection in a given case. In most cases chronic middle-ear suppuration is complicated not only by the cerebellar abscess, but also by suppuration of the labyrinth, circumscribed meningitis, or sinus thrombosis, affections which in themselves are of a serious nature. This is aggravated by the long duration of the initial stage. During this period the nutrition of the patient has considerably suffered, so that the operation must be performed upon debilitated, under-nourished individuals with slight power of resistance.

The operation itself is a serious matter, especially if the opening of the posterior cranial fossa is to be combined with resection of the labyrinth or with opening and evacuating the sinus sigmoideus. The after-treatment is also a difficult one, there being danger of pus descending to the bottom of the posterior cranial fossa, with consequent diffuse purulent meningitis. This danger exists for weeks after the operation. Even in favorable cases there is danger of a permanent cerebellar prolapse. No doubt, this could be removed in part and replaced by skin plastic, but this is always accompanied by the danger of a fresh abscess if the first abscess has healed with a cystic scar. There is also danger of diffuse meningitis setting in years afterward.

The mortality of 40 per cent. is based on all the cases that have come under my observation. It also includes those cases in which cerebellar abscess was not diagnosticated and death occurred after perforation of the abscess and suppurative meningitis.

The comparatively most favorable prognosis is offered by abscesses that have been operated upon in the latent stage. They are usually discovered at the time of a radical operation carried out during that period when the operative findings have rendered the exposure of the

posterior cranial fossa necessary, or a complicated suppuration of the labyrinth has rendered a resection of the labyrinth necessary. If under these circumstances the dura of the posterior cranial fossa has been exposed, a spontaneous evacuation of the abscess through a dural fistula into the operative cavity may terminate favorably.

LUMBAR PUNCTURE AND ITS SIGNIFICANCE IN OTOLOGY

Lumbar puncture, which was recommended in 1891 by Quincke as a diagnostic and therapeutic measure, has been adopted in otology.

Technic.—The instrument used is a hollow needle, the short pointed end of which is ground in the shape of a trocar. It is provided with a stylet, at the end of which there is a mark which will enable the operator to recognize the position of the stylet when introduced. The needle is closed with a metal stopper. The needle is from 8 to 14 cm. long and from 0.6 to 1.5 mm. wide. It is advisable to have in readiness several needles of different dimensions. The patient lies on one side with legs flexed and drawn up, so that the elbows will approach the knees as closely as possible. The spinal cord will thus bulge out convexly. The needle is introduced about 1 cm. away from the median line between the third and fourth or the fourth and fifth lumbar vertebræ. As the infantile conus medullaris is located at the level of the third lumbar vertebra, there is no danger of injuring the medulla. In order to find the correct point of insertion, it may be convenient to connect the highest points of both iliac crests by a straight line which will traverse the spinous process of the fourth lumbar vertebra.

A slight resistance will be felt, when the needle is introduced correctly, in penetrating the layer of muscles and ligaments. This resistance will cease as soon as the needle has advanced to the vertebral canal. The needle is firmly fixed if inserted correctly; if, however, it can be moved to and fro in the tissue, the right way has not been found. As soon as the operator is under the impression that the point of the needle has penetrated into the vertebral canal, the stylet is withdrawn and the exuding liquor caught in three sterile test-glasses. While passing from one glass to the next, the needle is closed with a metal stopper, so that all the fluid may be saved for the examinations. Under ordinary pressure conditions, 10–15 c.c. should be secured. With reduced pressure, the quantity withdrawn should not exceed 8–10 c.c.; with increased pressure, 30–50 c.c. may be evacuated without danger. The needle is then rapidly withdrawn and the puncture immediately closed with an iodoform gauze plaster.

The proceedings are best conducted under ether inhalation. Should a blood-vessel be injured, there may be pure blood at first, and the first portion of cerebrospinal fluid will still have an admixture of blood. This

should be caught in a special test-glass, as the fluid used for examination may not contain any admixture of blood. In some cases the puncture will give entirely negative results with the patient in the lateral decubitus. The experiment may then be repeated in the sitting or gibbous posture. Aspiration, in the event of there being no spontaneous evacuation, is to be strongly deprecated. The cause of a negative result may be faulty technic, pathological conditions in the vertebral canal or cerebral cavity, such as occlusion of the foramen Magendie, viscid, purulent secretion in the vertebral canal, circumscribed spinal meningitis with agglutination or adhesions of the soft meninges, or temporary negative pressure in the vertebral canal. Untoward consequences have never been reported in auricular or intracranial otitic affections, provided the puncture has been carried out with care. The puncture is, of course, contraindicated when the affection is complicated by a disease in which the puncture would always involve danger, as in diabetes, uræmia, cerebral tumor, or injury to the skull.

Clinical Examination of the Cerebrospinal Fluid

(1) *Pressure.*—In order to measure the pressure under which the fluid escapes, many authors have recommended the use of a manometer. The use of auxiliary apparatus, however, always includes the danger of infection, and, besides, the pressure varies considerably during the operation and greatly depends upon accidental causes, such as position of the body and cardiac function. A manometer may, therefore, well be dispensed with, and I content myself with estimating the pressure according to whether the fluid escapes in a strong gush, in an arch, slowly, or in driblets. Increased or considerably decreased pressure always points to considerable pathologic changes. In meningitis the pressure often remains unchanged.

(2) *Color.*—Normal cerebrospinal fluid is colorless. Yellow discoloration occurs in chronic non-suppurative meningitis, in arteriosclerosis of the cerebral arteries, in epilepsy and paralysis. Red discoloration occurs in pachymeningitis hæmorrhagica, in cerebral or ventricular hemorrhage, and in accidental admixture of blood from injury to the blood-vessels during puncture (see above). In the absence of any purulent inflammation of the brain or spinal cord the following rules hold good for the differential diagnosis, whether the admixture of blood be accidental or pathological:

In pathological admixture the blood is deposited at the bottom of the undisturbed test-tube without coagulation. In admixture of blood from an accidentally injured vessel there will be coagulation of the deposit. Considering that, in many cases of meningitis, diagnostically valuable coagulation occurs due to the inflammatory process itself, it

is clear that fluid containing accidental admixture of blood is of no value for deciding the question as to whether an affection is meningitis or not. For this reason the fluid should be caught in several tubes, so as to obtain pure fluid in the end. Should this prove impossible, another puncture may be made at a different place, or repeated on another day. There is hardly ever a question of hemorrhagic processes in otitic cerebral affections, so that, in the event of a blood-stained fluid appearing in otitic endocranial processes, an accidental admixture of blood may always be assumed. This can also be recognized by the discoloration becoming less intense, or the fluid becoming clear, with subsequent withdrawals either at the original or a new point of puncture.

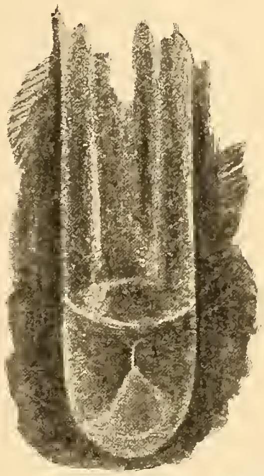
Fig. 117.

Minute, cobweb-like coagulations of spinal fluid in tuberculous meningitis in a one-year-old child.

Fig. 118.

Typical columnar coagulation with adhesive threads at the top and bottom of the test-tube, in suppurative diplo- and staphylococcus meningitis.

Fig. 119.



Conical coagulation ending in a point, in suppurative meningitis (streptococcus pyogenes).

(3) *Transparency.*—Normal cerebrospinal fluid is as clear as water. Gray, yellow-gray, or yellow-green discoloration is always a sign of purulent meningitis. In serous meningitis and in many cases of the tuberculous form the fluid is perfectly clear.

(4) *Coagulation.*—In meningitis there are fibrinous coagulations in the course of six to twenty-four hours in the undisturbed test-tube; in tuberculous meningitis they are exceedingly fine and in the shape of a cobweb permeating the entire fluid (Fig. 117), or they can be recognized as minute white specks suspended in the fluid or deposited at the bottom. In suppurative discoloration of the fluid the coagulation in the test-tube is either in the shape of a column (Fig. 118) or of a cone (Fig. 119), the column often inserted in threads both at the surface meniscus and at the bottom of the tube. Upon adding sodium citrate or oxalic acid to the freshly withdrawn fluid there will be no coagulation.

(5) *Chemical Changes.*—Normal fluid contains albumen only in traces. It contains no sugar, or only in traces (0.06–0.09 per cent.). An increased percentage of albumen is often found in cases of meningitis and cerebral tumors (1–2 per cent.). In tumors especially of the posterior cranial fossa the sugar content may be considerably increased. In diabetic coma aceto-acetic acid is sometimes found in the cerebrospinal fluid.

(6) *Cytology.*—The sediment, obtained with or without centrifuging, is put upon the slide by means of a pipette, and fixed by heat or alcohol ether. Staining is done with tri-acid, hæmatoxylin eosin, or after Romanowsky. The cytological examination should be made immediately after puncture and before coagulation. Should the latter have set in, the fluid is shaken with glass pearls until a uniform turbidity, or nearly so, has been obtained. The centrifuged sediment of normal cerebrospinal fluid contains only isolated lymphocytes.

In suppurative cerebrospinal meningitis there are polynuclear and large mononuclear leucocytes, together with normal white blood-cells. The lymphocytes in tuberculous meningitis are of particularly small size. Lymphocytosis is also present in all syphilitic and metasyphilitic affections of the brain and spinal cord; in the cerebral and spinal meninges likewise (paralysis, tabes).

Flaky blood pigment is a sign of old hemorrhages (cerebral trauma, pachymeningitis hæmorrhagica).

(7) *Bacteriology.*—In suppurative meningitis the pathogenic factors should be demonstrated first in the microscopic preparation, after which they should always be verified as pathogenic by cultures and animal experiments. It is not a rare occurrence for staphylococci, Gram-positive cocci, or diplococcus intracellularis to be microscopically demonstrable and yet to prove negative both in cultures and animal experiments. These are cases of impurities imparted to the fluid, especially when staphylococci are present, or bacterial impurities of the staining fluid, or of cerebrospinal fluid containing degenerated bacteria which are no longer pathogenic. In many cases of intracranial otitic affections where the cerebrospinal fluid is gray or turbid from pus, the fluid proves perfectly free from micro-organisms. These cases of purulent non-infectious meningitis are found in imperforate cerebral abscess and in extensive pus foci of the external dural surface (extradural abscess, ichorous sinus thrombosis). Again, in the early stages of suppurative infectious meningitis, the cerebrospinal fluid may be still sterile, while a later puncture will yield fluid containing pathogenic micro-organisms. In circumscribed purulent meningitis the fluid may remain perfectly clear and sterile for a considerable time. It is only in exceptional cases that clear non-bacterial fluid is observed in the early stages of circum-

scribed suppurative pachymeningitis and intrameningeal abscess. In tuberculous meningitis it is advisable to make a bacteriological examination of the cobweb coagula. If spontaneous coagulation does not occur, or if it is inconvenient to wait for it, a small piece of cotton wool may be placed in the liquid, as this will be rapidly surrounded by coagulations. According to Ziehl, tubercle bacilli can be demonstrated in about 75 per cent. of all tuberculous cases, but it may be necessary to prepare a large number of specimens. If the microscopic demonstration is unsuccessful, animal experiments should be resorted to, according to Bloch. When using guinea-pigs, the best way is to injure the inguinal lymph-glands by crushing. This will predispose them to tuberculous infection. A few c.c. of the cerebrospinal fluid to be tested are injected into the glands, which in positive cases will show tuberculous infection in one or two weeks.

The following table will illustrate the total findings of cerebrospinal fluid both in normal cases and in the various forms of otitic meningitis:

CEREBROSPINAL FLUID

Diagnostically important properties of cerebrospinal fluid	Endocranium normal	Serous meningitis	Suppurative non-infectious meningitis	Suppurative infectious meningitis	Tuberculous meningitis	Admixture of blood
Pressure	Normal	Increased	Normal or increased	Normal, elevated or decreased	Normal	Normal
Color	Colorless	Colorless	Gray	Gray or yellow	Colorless or gray	Reddish
Transparency	Clear	Clear	Turbid	Turbid	Clear or turbid	Turbid
Coagulation	None	Present	Present	Present	Present	Present
Cytological findings	L	L	LP	LP	LP	LE
Bacteriological findings	Negative	Negative	Negative	M	Tbc	Negative

L—Lymphocytes. P—Polynuclear leucocytes.
M—Micro-organisms. E—Erythrocytes.
Tbc—Tubercle bacilli.

The high diagnostic value of lumbar puncture is indisputable, as it furnishes the only reliable means of learning the exact condition of the meninges in a given case. Its indicational value, however, is only of subordinate importance, as an operation cannot be regarded as devoid of success even when the cerebrospinal fluid shows the gravest changes and demonstrates the presence of infectious, purulent, diffuse meningitis. A contraindication to operation can only be found in the general manifestations, an unfavorable condition of the heart and respiration, but never in the findings of lumbar puncture. Besides, operation in all these cases is urgent, so that it would be out of the question to wait for the culture or animal test. In most cases lumbar puncture is carried out as a preliminary act to the operation or immediately afterward.

The idea of abstaining from an operation just because the spinal fluid is turbid from suppuration has been entirely discarded.

Lumbar puncture is employed as a therapeutic measure in the after-treatment of operative cases of otitic meningitis, and many authors have applied it with some degree of success in non-suppurative affections of the internal ear, in cases of obstinate subjective noises or vertigo. In these cases the puncture is best carried out under the influence of ether inhalation, and repeated at not too frequent intervals according to the nature and course of the affection.

Ventricular puncture can be best carried out after operative exposure of the middle cranial fossa in cases of intracranial otitic affections where lumbar puncture has not yielded any fluid or not a sufficient amount of it.

Ventricular puncture is contraindicated if perforated cerebral abscess is suspected.

Lange in 1912 demonstrated that the action of cerebrospinal fluid in various conditions upon a colloidal gold solution could be used as a delicate test, differentiating normal from pathological cerebrospinal fluids, and more particularly syphilitic from other affections of the central nervous system.

The theory of the reaction is based upon the following observations made by Zsigmondy in the course of his study on metallic colloidal solutions:

1. Solutions of electrolytes precipitate colloidal gold.

2. Proteins in the absence of an electrolyte also precipitate a solution of colloidal gold.

3. Proteins in the presence of an electrolyte inhibit precipitation in colloidal gold solution, the so-called "Gold-Schutz."

The relation existing between this opposed reaction of electrolyte and protein is definite for the same protein but differs when a different protein is used, and is therefore a specific property of the individual protein.

Lange's application of these principles to the study of spinal fluids is based upon the theory (1) that substances in pathological spinal fluids will precipitate colloidal gold provided the globulin and nucleoprotein fractions are held in solution with a 0.4 per cent. sodium chloride solution, and (2) that there is a characteristic change for certain diseases involving the central nervous system.

The test is a delicate one and its success depends upon the use of scrupulously clean glassware, accuracy in all measurements, and avoidance of bacterial contamination. The presence of blood or serum vitiates the findings.

XIV. TRAUMATIC INJURIES OF THE ORGAN OF HEARING[1]

Isolated traumatic injuries of the chain of auricular ossicles are only seldom observed. These are caused almost exclusively by foreign bodies in the auditory canal which have advanced into the middle ear by unsuccessful attempts at extraction. Aside from rupture of the tympanic membrane, there may be luxation of the auricular ossicles, fracture of the neck of the malleus, tearing of the chorda tympani, hemorrhages of the middle ear, and inflammatory changes in the region of the middle ear.

Traumatic fissures and fractures of the temporal bone in childhood are of the greatest rarity, owing to the great elasticity of the cranial bones.

The cause of acquired deafness is often stated to be a fall on the head, but the exclusive cause is meningitis. It may be that the child fell out of bed during convulsions, landing on his head, or that the symptoms of cerebrospinal meningitis had not previously been noticed by the parents, and the fact of his suddenly falling down was erroneously regarded as the cause of the affection.

Fractures of the temporal bone in childhood are part manifestations of serious general injury, and will only occur under employment of considerable force.

The local signs of fissure and fracture in childhood do not differ from those of the adult. Fissures of the tegmen tympani lead to accumulation of blood in the middle-ear spaces (hæmatotympanum), and otoscopic examination shows black-red discoloration of the tympanic membrane which bulges strongly outward.

Fractures of the upper wall of the auditory canal and the tympanic roof lead to traumatic tearing of the membranous auditory canal. Examination shows effusion of blood from the external meatus. The tympanic membrane is usually covered by blood coagula and is not visible through the otoscope. If the fracture of the temporal bone has also caused tearing of the dura, there will be evacuation of cerebrospinal fluid through the external auditory meatus. In most cases fluid will only escape after the blood has been expelled, often as late as one or two days later. Fractures of the petrous bone which take their way through the labyrinth are apt to cause deafness rapidly with acute symptoms (labyrinthine vertigo, equilibrial disturbances, and vomiting). In a few cases facial and abducens paralysis have been observed.

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[1] The traumatic injuries of the external ear have been discussed on p. 111, those of the tympanic membrane on p. 128.

Course.—Fractures of the temporal bone through the labyrinth cause permanent deafness. Paralysis of the facial nerve is usually completely cured. The middle ear will undergo healing under gradual resorption of the blood, provided no secondary infection occurs. Should such occur, there will be suppurative disintegration of the coagula accumulated in the middle-ear spaces and suppurative otitis media. Complication by traumatic meningitis renders the prognosis very unfavorable.

Treatment should include rest in bed and application of ice-bags to the head. In examining the ear nothing should be done but carefully removing the coagula from the auditory canal and applying a bandage over the ear. Any other manipulations should be refrained from, such as syringing, air insufflation, etc. If vertigo should persist, the auditory nerve should be treated with the galvanic current, producing galvanic nystagmus in the opposite direction to the prevailing pathologic one.

In the light forms of traumatic concussion of the labyrinth following a direct or indirect injury to the skull, there is usually some difficulty of hearing. Manifestations of the static labyrinth may be entirely absent or consist in slight spontaneous nystagmus without vertigo. The prognosis is favorable, and after a short time recovery usually takes place with perfectly normal hearing ability.

In cases of serious traumatic concussion of the labyrinth there is considerable impairment of hearing, or deafness; in most cases there are also pronounced irritative manifestations of the static labyrinth, consisting in vertigo, equilibrial disturbances, and vomiting, which will gradually disappear. Healing in the region of the static labyrinth often occurs with maintained reflex excitability of the labyrinth, its complete loss being rare. Deafness will persist unchanged. Where hearing ability is preserved, the cases show a different behavior; in some few the hearing acuity is improved in the course of time, in others the degree of impaired hearing remains constant, and in a third group the difficulty of hearing gradually increases and may lead to complete deafness.

XV. MALIGNANT NEW-FORMATIONS OF THE EAR

Carcinoma of the auricular region is extremely rare in children. I have observed a single case, where a young man sixteen years of age had inoperable carcinoma of the middle ear. This undoubtedly started from the glands of the auditory canal and developed, like all middle-ear car-

Fig. 120.

Sarcoma of the external ear and middle ear, starting from a small spindle-cell sarcoma of the parotid. Girl, fourteen years of age.

cinomata, from a chronic suppuration of the middle ear. I saw the patient at a time when the entire temporal bone was infiltrated by carcinomatous masses, the entire auricular region bulging out convexly, and the carcinoma had caused paralysis of the facial nerve through erosion of the capsule of the labyrinth. Leideler observed a middle-ear carcinoma in a young man of nineteen.

Sarcoma of the ear occurs less often in childhood. These are either cases of subacute leukæmia which have developed a lymphosarcoma, originating at the cervical glands, destroying the base of the petrous bone, and penetrating into the middle ear or external auditory meatus, or cases where the tumor has developed from a tonsillar or parotid sarcoma (sarcomatous mixed tumor, small-celled round- or spindle-cell sarcoma; Fig. 120). Sarcoma of the ear emanating from the dura is very rare in youth.

Course.—Sarcoma causes in a very short time fetid suppuration of the middle ear, extensive ulcerations of the external auditory meatus, and paralysis of the facial nerve. Unless the original affection leads to death, the latter will in most cases be due to suppurative meningitis consequent upon spreading of the suppurative middle-ear affection to the endocranium, or owing to loss of blood consequent upon erosion of the carotid, sinus sigmoideus, or bulbus jugularis.

The prognosis is unfavorable in all cases.

Treatment.—The conservative treatment of sarcoma consists in careful exposure to the X-ray or radium, in regard to which an experienced X-ray specialist should be consulted.

Operative interference is contraindicated in leukæmic or lymphosarcomatous tumors, as the operative trauma and after-treatment only seem to hasten the end. Sudden occurrence of laryngeal stenosis in case of tonsillar sarcoma may render tracheotomy necessary. Sarcoma which has originated from parotid tumors is the only form which admits of surgical interference as long as the tumefaction is confined to the external ear and the middle ear is perfectly intact; but even in these cases the chances of permanent recovery are small.

In the rest of the cases there is nothing to be done but resort to symptomatic general treatment, and local treatment of the fetid middle-ear suppuration which will set in as soon as the tumor spreads to the middle ear.

The patient must be made comfortable, especially in later stages of the disease, by morphine. Lysol irrigation does much to control the disagreeable odor of the necrotic bone.

XVI. AFFECTIONS OF THE EAR IN GENERAL DISEASES

I. AFFECTIONS OF THE EAR IN DISEASES OF THE BLOOD AND THE BLOOD-FORMING ORGANS (LYMPHOMATOUS EAR DISEASES. AFFECTIONS OF THE ORGAN OF HEARING BY LEUKÆMIA, CHLOROMA, AND KINDRED DISEASES)

Lymphomatous ear diseases and their pathological manifestations are principally favored by the hemorrhagic diathesis. Hemorrhages of the ear occur in a large number of cases, especially in those where hemorrhages also occur in other parts of the body. Bleeding from the ear occurs particularly in those forms of acute lymphomatosis which are apt to develop from acute hyperæmia of the auricular region, the base of the skull, or the entire head. Bleeding of the middle ear leads to hæmatotympanum and, owing to bacterial infection, to hemorrhagic suppuration of the middle ear under considerable elevation of temperature.

Circumscribed hemorrhages of the labyrinth lead to complete deafness and loss of reflex excitability of the labyrinth, either by way of various single attacks or without any warning. Apoplectiform hemorrhages of the labyrinth may lead to destruction of the entire neuro-epithelium. Sudden effusion of viscid blood from the labyrinth may lead to compression of the membranous labyrinth; slow effusions, often repeated, to partial obliteration of the membranous labyrinth. Hemorrhages have also been observed in the internal auditory canal and along the nerve-sheaths of the auditory and facial nerves. Blood effusions of the aqueducts or internal auditory canal destroy the normal lymph circulation of the internal ear, which may again lead to ectasia of the membranous labyrinth. Continued bleeding of the labyrinth causes secretion of large quantities of pigment in the labyrinth.

Lymphoid infiltration is found in the tympanic cavity and the mucous membrane of the tube, and often in the perilymphatic connective-tissue layer of the internal ear, sometimes in the stria vascularis, in the ligamentum spirale, in the crus cerebri, and the root region of the octavus.

The inflammatory changes consist in small-celled, apparently lymphomatous infiltrates, which later disintegrate by suppuration and are permeated by micro-organisms.

Acute exudation of the labyrinth is only found in the early stages of hemorrhagic labyrinthitis.

In cases of prolonged duration secondary changes of the labyrinth and middle ear may occur in the shape of pathological connective-

tissue and bone proliferations, and degenerative atrophy of the sensory epithelium and of the peripheral and central branches of the auditory nerve. I have also found lymphoid tumefaction of the labyrinth in cases of leukæmia in the presence of lymphosarcoma and chloroma. Hyperæmia of the ear is exceedingly common in lymphomatosis. Anæmia of the ear, notably of the labyrinth, is the result of connective-tissue obliteration of blood-vessels following hemorrhages of the labyrinth or after displacement of regional small arteries through lymphoid tumors.

The diagnosis usually offers no difficulties. In all cases of hemorrhagic otitis or when hemorrhage of the labyrinth is suspected it is advisable to examine the blood microscopically.

Treatment.—There can, of course, be no other treatment but symptomatic. Hemorrhage from the external meatus should be thoroughly wiped out with cotton tips and controlled by insufflation of iodoform powder and insertion of iodoform gauze. The same applies to hemorrhagic otitis. The treatment of inflammatory changes of the tympanic cavity and mastoid does not differ in any way from that in non-lymphomatous forms. In mastoid affections the possible necessity of surgical interference should not be lost sight of.

Transitory improvement sometimes sets in spontaneously. The prognosis of lymphomatous affections of the internal ear, and particularly that of apoplectiform deafness caused by hemorrhages, is unfavorable.

II. CONSTITUTIONAL EAR DISEASES

1. AFFECTIONS OF THE EAR IN LYMPHATIC CONSTITUTION AND RHACHITIS

So far as the affections of the external ear are concerned, mention may be made of obstinate eczema of the concha and external auditory meatus. Lymphatic individuals very frequently suffer from catarrhal affections of the middle ear. Suppurative otitis media is also often observed. In this class of patients there is a tendency to develop a chronic state of both catarrhal and suppurative middle-ear affections. Older lymphatic children often suffer from neurasthenic troubles of the ear, disagreeable subjective noises, or hallucinations of hearing. Some of them are much molested by the subjective noises (humming) of the cervical veins, which are often unilateral, but may be bilateral.

General invigorating treatment is of the greatest importance in conjunction with local treatment.

Owing to impaired communication between periosteum and bone, subperiosteal and extradural abscesses may insidiously develop in lymphatic individuals and attain a considerable size. Subperiosteal abscesses of the mastoid may under these circumstances extend to the vertex and middle of occiput, or even beyond the median line to the

other cranial hemisphere. Owing to perforation of abscesses through the osseous wall of the auditory canal, the membranous part of the canal may be completely bathed in pus, and the suppuration may spread to the submaxillary articulation and the soft parts of the mouth.

Among the intradural abscesses in lymphatic patients, the very extensive abscesses at the base of the posterior cranial fossa and the large abscesses in the projection of the tympanic roof and the temporal squama are most known and dreaded.

In rhachitic children there are characteristic arrests of development of the temporal bone. The cortical layer is especially thin and porous. The fissura mastoidea of older children may still contain cartilaginous remnants corresponding to the lateral wall of the antrum. In suppurative inflammation there is danger of a rapid spreading of the suppuration to the bone and rapid suppurative resorption of the latter. In suppurative inflammation of the antrum there is rapid perforation outward, with formation of a subperiosteal abscess of the mastoid region. The petrous bone may still have an exceedingly tender and apparently diploic structure in advanced childhood, so that middle-ear suppuration in rhachitis may lead in an incredibly short time to a suppuration of the petrous bone, either with paralabyrinthine pus foci or necrosis of the petrosis bone and labyrinth.

2. DISEASES OF THE EAR IN ENDEMIC CRETINISM. ENDEMIC CONSTITUTIONAL DYSACUSIS (DIFFICULTY OF HEARING) AND DEAFNESS

The following statements are based upon the experience I have gathered in the study of the material collected and treated by von Wagner. This material comprises the district of Judenburg in Styria, where endemic cretinism is particularly rampant. I commenced my investigations in 1904 and since then have uninterruptedly continued.

Occurrence.—Difficulty of hearing, or dysacusis, is exceedingly frequent in cretinism. Examination of considerable material has shown that hardly one-fourth of all cretins are endowed with normal hearing acuity. In most cases there is a medium reduction, in 20–30 per cent. of those partly deaf there is a high degree of reduction, and in about 5 per cent. there is complete deafness.

The ear affections of most cretins are congenital or have existed from earliest infancy. In other cases they develop, like the other cretinic symptoms, after acute infectious diseases and other serious general affections, or at least become more accentuated than before, or they occur without such additional factors. Cretinic deafness, however, is always congenital, as are all the other cretinic signs of a grave nature.

Bircher proposed to designate as "endemic deafness" that form of deaf-mutism which is peculiar to endemic cretinism and frequently

characterizes it as apart from all other forms of deafness. Bircher's endemic deaf-mutism corresponds with Hammerschlag's endemic constitutional deafness. Bircher points out the various degrees of physical and mental development in deaf-mute cretinism, stating, "There are such as are well developed in all respects aside from the absence of hearing and speaking, while others show all degrees of physical and psychic decadence."

Bloch established the conception of "dysthyral deafness," based upon the material he collected. He emphasizes the frequency of medium reduction of the acuity and of lingual disturbances. He also mentions the favorable effect of thyroid feeding upon the deafness of cretins.

Siebenmann attacks the conception of "dysthyral" deafness, stating that in a case of total aplasia of the thyroid the ear and particularly the labyrinth were anatomically intact. According to my opinion, this proves nothing. Among the large material I have examined, I have here and there found fairly good intelligence and good hearing acuity, although there was no demonstrable thyroid; in some of these cases, at least, the labyrinth was intact. True, Bloch has found changes of the labyrinth in a far greater number of cretins than myself, and admits that dysthyral difficulty of hearing is not very frequent or even always considerable. He refers to cases of impaired hearing of the lightest degree in which hypothyroids never were conscious of their defect and where the latter could only be detected by careful examination of the ear. Possibly Bloch includes in his nomenclature various pathological forms which do not belong together either anatomico-pathologically or etiologically. Nevertheless, Bloch's conception may be accepted, led by the impression gained from the observation of considerable cretinic material, and in view of the fact that there are very many goitrous cretins with defective hearing and many cases of considerable dysacusis and deafness in hypo- and athyroid cretins.

Kocher assumes a central seat of deafness and speaks of an auricular sensory aphasia.

Von Wagner distinguishes between certain types of cretins. The dwarfish cretin is characterized by the presence of all cretinic symptoms: dwarfish structure, high degree of myxœdema, complete absence of sexual development. In these cases there is hypothyroidism, there being only a small atypical goitrous nodule or even no trace of that. This type includes many cases of fully developed idiocy, dysacusis, or complete deafness. Cretins without any appreciable hearing defect and with good intellect are rare in this class.

In other types of cretinism the principal sign is endemic deaf-mutism. There are serious disturbances of the organ of hearing or total deafness, together with considerable impairment of psychic functions.

Among the dwarfish half-cretins slight affections of the middle ear and labyrinth are not infrequent, while serious disturbances or deafness do not exist.

In the type of the cretinic dwarf characterized by dwarfish stature, myxœdema, and infantile genitals, there are no appreciable disturbances of intellect or hearing.

A very frequent type of cretins comprises those with a very pronounced goitre. In these cases, in which all other cretinic signs are overshadowed by the goitre, there is hardly one with normal hearing, but complete deafness is rare. This group includes both half-cretins and full cretins, cases with slight and with considerable hearing defects; deaf-mute cretins and half-cretins likewise belong to this group. The last type is characterized by deaf-mutism and goitre. In these cases there is no arrest of growth, no cretinic expression, no myxœdema, and no defective genitals. Nor is their intelligence materially disturbed, but there is a certain degree of mental dulness and heaviness in nearly all cases.

Anatomy.—Catarrhal changes of the middle-ear spaces are the cause of dysacusis in a large number of cases. Considerable enlargement of the faucial tonsils and chronic thickening and swelling of the nasopharyngeal mucosa are exceedingly frequent in cretins. The catarrhal changes of the middle ear originate from the nasopharyngeal changes, typical forms of exudative middle-ear catarrh being only found exceptionally. The frequency of middle-ear affections in cretins is also dependent upon the fact that the myxomatous swelling of the nasopharyngeal mucosa is communicated to the mucosæ of the tube and tympanic cavity.

Moos has found bilateral hyperostosis of the posterior and interior wall of the tympanic cavity in a case of idiocy (fetal chondrodystrophy).

Hammerschlag found in a dwarfish girl reduction of the left middle-ear spaces and a rigid connection between the stapes and incus. The shanks of the latter were coarse and the plate was reduced to one-third of the normal size.

Siebenmann, in a case of aplasia of the thyroid which he examined anatomically, found no changes that could have occasioned any material functional disturbance of hearing. In another case Siebenmann found Corti's organ to be in a lower position than usual, while the tunnel space was particularly large.

Habermann noticed arrest of development in Corti's organ and displacement of the cells of the spiral ganglion.

Manasse demonstrated changes of the auricular skeleton and membranous labyrinth. He looks upon the changes of the bone as primary and congenital, and those of the soft parts as secondary.

Suppurative inflammatory changes of the middle-ear spaces are rather rare in cretinism. Those few cases of chronic middle-ear suppuration which I have observed belong to the group of suppuration following acute infections. In these cases the middle-ear inflammation had set in after scarlet fever, measles, and diphtheria.

The changes of the internal ear consist in many cases in degenerative atrophy of the auditory nerve and the nerve-endings of the labyrinth. In some cases the nerve terminations of the static labyrinth as well as Corti's organ are involved in the atrophic process. In other cases the atrophy is restricted to Corti's organ. The latter form of affection approaches the type of sacculocochlear degeneration (degeneration of the pars inferior) in congenital deafness. In high degrees of cretinic dysacusis it is a typical occurrence to find the corner of the cochlear window filled with mucoid connective tissue or fatty tissue. This tissue embolus extends at one side to the tympanic mucosa and at the other side to the membrane of the cochlear window. Sometimes the corner of the vestibular window and the lumen of the stapes are also replete with mucous connective tissue.

It is, of course, to be expected that in congenital deafness, representing as it does the principal type of endemic constitutional deafness and dysacusis, the anatomical findings should vary considerably. In one of the cases of highly developed dysacusis which I observed, the capsule of the labyrinth contained pathological osseous foci of the type of osteitis vasculosa, such as is found in otosclerosis. There was also degenerative atrophy of the eighth nerve. In many cases the anatomical cause of deafness seems to be a more or less complete obliteration of the membranous labyrinth with aplasia of the nerve-end places and a stunted eighth nerve.

I have found partial obliteration of the cochlear canal in a cretinic child with a high degree of partial deafness. The hair-cells of Corti's organ were completely absent, and the pillar cells partly so.

Degeneration of the stria vesicularis seems to be a characteristic sign in nearly all cases of pronounced hardness of hearing and deafness in endemic cretinism. In one case there were epithelial duplicatures and septum formation of the membranous cochlear canal of the type of septum found in dancing mice, in the congenitally deaf and undeveloped albino cats and dogs.

The connective-tissue portions of the labyrinth are considerably atrophied in some cases; even the ligamentum spirale of the cochlea may be atrophied to such an extent that only a few fibre remnants remain. In the same cases, however, there is often considerable connective-tissue proliferation of the canalis spiralis, so that the atrophied spinal ganglion seems surrounded by a thick connective-tissue capsule.

One case has come under my observation in which there was considerable thickening of the membranous walls of the vestibule and especially of the external wall of the utriculus.

In the cases of cretinic deafness which I had an opportunity of examining anatomically there were no pathological changes in the region of the auditory nuclei, in the central ramifications of that nerve, or in the cortex of the temporosphenoidal lobe. The peripheral, highly atrophied eighth nerve possesses perfectly normal nuclei in the crus cerebri and fibrous bundles running in a central direction. There is, however, still a possibility of auditory disorders being caused by changes of the central auditory nerve in isolated cases.

Slight abnormalities of the concha, such as unusually large or small lobules, flat or kinked helix, Darwin's macacus point, deflected ear, asymmetric position of the concha, are fairly frequent in cretins, but I have not observed any gross malformations. The external auditory meatus was always normal. Nor have I found any exostoses.

Symptoms.—The nasopharyngeal changes in cretinism lend themselves to a division into three groups of symptoms. The first group deals with reduced hearing acuity, the second group with abnormal respiration, and the third group with abnormal articulation and lingual and mental insufficiency.

The psychic behavior of cretins is important for the interpretation of the auditory disorders. The cretinic apathy, physical and psychic hebetude, may lead one to suspect considerable impairment of hearing which in reality does not exist. In the highest degrees of cretinism there may be no reaction whatever to a sound although the auditory apparatus may be perfectly in order.

Subjective noises are only exceptionally complained of; the only cases I have observed referred to older cretins with but slight cretinic habitus, good intelligence, and slight catarrhal affections of the middle ear. There were only two cases of labyrinthine vertigo. In one of them there was deafness after cerebrospinal meningitis in a child; in the other there was medium hardness of hearing.

The tympanic membrane presents more or less important catarrhal changes in most cases. It has a dull appearance, is discolored gray or reddish gray, and shows all degrees of retraction. Lime deposits in the tympanic membrane and accumulations of exudate in the middle ear are rare. I have not often met with suppurative inflammatory changes of the middle ear: there is nothing characteristic for cretinism in them, and they do not differ in any way from those in otherwise normal individuals.

Functional Findings and Functional Tests.—In light degrees of cretinism the functional tests do not differ in any way from those made in

normal children. Reliable results with the tuning-fork can, therefore, not be expected before the eighth or tenth year, especially if an exact and complete tuning-fork test is required. In the presence of important psychic disorders an exact functional test is impossible. We will then have to be satisfied with establishing the most important points (length of perception by bone-conduction, watch-ticking through the cranial bones, perception of c^1-fork) in order to recognize any possible affection of the labyrinth. If there is only rudimentary reaction to acoustic impressions, the decision will be limited to the question as to whether or not there is any hearing ability.

Greatly impaired intelligence in cretins with normal hearing may erroneously cause the impression of considerable partial or complete deafness.

The aphonia of many cretins capable of hearing may be designated as aphasia or psychic deafness. These are not usually cases of pure cretinism, but combinations of cretinism and idiocy, and aphonia in these cases is not only caused by psychic impairment, but also by cerebral changes.

Development of speech may be completely arrested owing to psychic insufficiency, or remain on a very low level in spite of good hearing ability. In cases of this kind the functional test may be a complete failure, as, owing to his hebetude, the cretin will fail to react to the loudest noises, in spite of good hearing ability.

In most of these cases, however, the family, and especially the mother, will be able to impart useful information, stating, for instance, that the child would like to speak, but cannot. Continued observation will also conduce to a better understanding of their faculties. Thus, there may be noticed a motor reaction to the bark of a dog, the sound of a trumpet, rattle, etc., when the child believes himself unobserved.

An important aid in deciding upon the hearing ability of cretins is their gait. Cretins who are very hard of hearing or totally deaf have a gliding, grating, groping gait. Walking with extended legs is found only in cases where the static labyrinth is out of function. I have observed spontaneous nystagmus in no more than five cases, in two of which there was degenerative affection of the labyrinth, while the others were highly nervous, restless cretins who, besides, suffered from epileptic convulsions. The hearing acuity for conversation and whispering can only be determined in cretins of fairly good intelligence. Unintelligent cretins, even with good hearing ability, will not repeat the test words at all or only indistinctly, or else they will give the same stereotyped answer to widely different test words. In making a functional test on cretins with inferior intelligence, no questions in regard to sounds or noises should be asked to which the answer is simply yes or no. Even

intelligent cretins are easily susceptible to suggestion, and the less intelligent ones will mechanically reply "yes" to each question for a time and then resort to a stereotyped "no."

The reflex excitability of the static labyrinth is perfectly normal in most cases. In some I have observed the reflex nystagmus produced after 10 rotations or by heat lasted for an unusually long time (30–40 seconds, as against the normal time of 15–20 seconds). But since in these cases there was neither spontaneous nystagmus nor any symptoms on the part of the static labyrinth, this increased reflex excitability cannot be regarded as pathological, but merely as a variety within normal limits. In some cases of cretinic deafness the reflex excitability of the static labyrinth was maintained; in others it was absent.

Diagnosis.—In cretins who respond well to the tests an exact diagnosis of the otitic affection can be made; in those incapable of intelligent response it will be necessary to be content with an approximate determination of the degree of dysacusis, or even with a decision as to whether the cretin is deaf or not. In three cases of adult cretinic idiots I was unable even to determine the latter question.

The characteristic hearing disorders of cretins may be clinically divided into three groups. The first group deals with catarrh of the middle ear, which in cretins is caused by hypertrophy of the lymphadenoid tissue of the nasopharyngeal tract, especially adenoid vegetations, far more frequently than in normal individuals. The second group comprises those cases of cretinic deaf-mutism which, barring exceptions, belong to the type of endemic deafness and deaf-mutism. To this group belong cases of pure cretinism as well as combinations of cretinism and idiocy. The third group refers to cases of medium hearing ability in comparatively intelligent cretins, in whom an exact functional test reveals an affection of the internal ear. The anatomical findings will show a degenerative atrophy of the octavus and the nerve-end places of the labyrinth.

It is a well-known fact that the psychic signs of cretinism sometimes appear in the wake of acute infectious diseases. It is not yet quite decided whether labyrinthine hardness of hearing of cretins is congenital or a sequel of acute infectious diseases. I incline to the former view, because in normal children these affections occur very rarely, and then almost exclusively after typhoid fever.

Moderate changes of the internal ear do not seem to be very rare in cretinism, but in view of the impaired intelligence the tuning-fork test is necessarily imperfect in most cases, rendering an exact localization of the otitic changes impossible.

Treatment.—The treatment of cretins with thyroid substance has found an ardent advocate in v. Wagner, who, as far back as 1904, em-

phasized the importance of the question as to whether it would be possible to remove the lingual disturbance and particularly the underlying hearing disturbance by thyroid treatment. This is really possible in a large number of cases. The thyroid tablets of Burroughs, Wellcome & Co. are to be recommended for this purpose. Each tablet contains 0.324 Gm. of active thyroid substance. Von Wagner prescribes in most cases 1 tablet a day, which may be temporarily increased to 1½–2 tablets if indicated. In weak infants, where a dose of 1 tablet may lead to disorders, such as acceleration of the pulse, excitation, convulsions, acute emaciation, depression, and disturbed sleep, the dose should be reduced to ½ tablet a day, or the medication should be interrupted altogether for a time.

The changes of the middle ear in catarrhal affections of that region depend upon the condition of the nasopharyngeal tract. Thyroid medication leads to a quantitative decrease of the lymphadenoid tissue and reduced swelling of the incrassated mucous membranes of the nasopharynx. This is followed by an improvement and sometimes by a complete cure of the middle-ear catarrh, with the consequence that the hearing acuity is improved or becomes normal.

The hearing disturbances of cretins which are caused by changes of the internal ear may sometimes be influenced by thyroid medication. There are several cretins among the cases I have observed whose dysacusis was definitely demonstrated and who experienced an improvement in their hearing acuity under the thyroid treatment instituted by v. Wagner. The duration of perception through the cranial bones had increased, c^4 was audible much longer, and the ticking of a watch which previously could not be heard was now perceived through the cranial bones. I have observed two of these cases for seven years, during which I found that the improvement set in comparatively rapidly, but after having reached a certain degree of perfection remained constant. The hearing acuity of these two cases has not changed for these five years, and in spite of thyroid treatment it has not been possible entirely to remove the changes of the internal ear. It is satisfactory to note, however, that there has been no relapse and that the improvement once attained has remained constant.

In cases of cretinic deafness administration of thyroidin offers no chance of success. Deaf cretins with good intelligence should be placed in an institute for the deaf-mute, otherwise in an asylum for imbeciles. V. Wagner is right in stating that the hebetude of cretinic deaf-mutes is usually so great that they are refused admittance by schools for the deaf-mute.

In aphonia (psychic, cretinic aphasia) thyroidin treatment has in most cases given surprisingly good results, the intellect having been

developed in many cases. As the latter increases, reaction to sound which was previously absent is incited, next follows development of speech, and after one or two years' treatment there is excellent hearing ability in cretins who before treatment seemed to be totally deaf. Unfortunately, these improvements soon come to a halt, and it would be a mistake to regard these cases as cured. The physical and mental development of these children had been considerably retarded, and their hearing ability which was unquestionably present was unable to assert itself, owing to their high degree of intellectual insufficiency. If children of this description are placed in institutions for the imbecile, they are usually taken for deaf. It requires attentive observation to recognize whether a child is capable of hearing, and his mental development has been retarded to such an extent that the hearing ability cannot assert itself, so that there can be no question of lingual development. In some cases there is nothing but rudimentary articulation even after thyroid treatment, while others will remain mute. In the former cases, however, the improved hearing ability is demonstrable by functional tests, and it is certainly worth noting that even in these cases the excitability for sounds can be awakened or improved by thyroid treatment.

Endemic deafness, likewise, is the object of successful treatment in cretinic degeneration. Von Wagner has instituted valuable attempts in this direction. In the district of Judenburg, Styria, where there are many families with positively cretinic children, he administered thyroid tablets to pregnant women and later to the new-born.

III. AFFECTIONS OF THE EAR IN ACUTE INFECTIOUS DISEASES

Acute infectious diseases in childhood endanger the hearing properties to a great extent. The development of otitis media is favored by swelling of the mucous membrane in the nasopharyngeal tract, by continuous passive dorsal decubitus, and by disturbed nasal respiration. The respiratory difficulties are particularly caused by voluminous accumulations of secretion in the nasopharyngeal tract and the inability of expectorating to a sufficient extent. This will cause irregular ventilation of the tympanic cavity after a short time. Subjective and objective signs of continued occlusion of the tube, such as sensation of fulness in the ear, heaviness in the head, moderate reduction of hearing acuity, are demonstrable in older children. Immigration of pathogenic germs from the nasopharyngeal tract into the tympanic cavity leads, directly or through catarrhal changes of the middle ear, to inflammatory and suppurative affections of the same.

Both the labyrinth and the auditory nerve are endangered by many affections in the course of acute infectious diseases, such as suppurative labyrinthitis, panotitis, neurolabyrinthitis.

Prostration of the little patient and insufficient nursing bring on the danger of necrosis of the auricular cartilage. This may be spontaneously produced by ischæmia in the condition of marasmus; otherwise it is due to kinking or wrong position of the concha while lying down on one side. The blood circulation in the concha, which in cases of great prostration is weak, will be entirely arrested through kinking, and necrosis, which will develop therefrom, can be recognized by the appearance of brownish-black spots.

These considerations are the foundation for some very important hygienic rules concerning the ear.

The ear should be carefully examined in all cases of acute infections of children, even if they do not complain of any otitic symptoms. Should the latter be present, otoscopic examination is particularly urgent if they can at all be referred to the ear in accordance with the symptomatology as described in previous chapters (decrease of hearing acuity, headache, vertigo, equilibrial disturbances, vomiting, etc.).

In order to prevent infection of the middle ear from the nasopharyngeal tract, instillations of a ½ per cent. solution of silver nitrate into the nose have been recommended by S. Weiss and ½ per cent. oily menthol solution by Gomperz. Disinfection of the oral cavity by rinsing or the use of formamint tablets (3–5 daily) is of importance in all cases.

Free respiration should be secured in seriously affected children, and care be taken by frequent changes of position that there be no accumulation of mucus in the nasopharyngeal space. Adrenalin plugs (sol. adren. 1.0, glycerin. aq. dest. āā 15.0) may be inserted into the nasal canals; this, and careful cleansing with cotton tips saturated with vaseline oil or a ½ per cent. menthol vaseline solution, will keep the same permeable. Care should be taken to have the concha in the correct position in the lateral decubitus, and it is often advisable to support it by a small cotton-wool cushion. The external auditory meatus should be firmly plugged with cotton wool previous to applying ointments or liniments to the head, especially if they contain highly irritative medicaments, or previous to douching the head or bathing.

1. AFFECTIONS OF THE EAR IN SCARLET FEVER AND MEASLES

Anatomy.—In typical cases of scarlatinal and morbillous otitis the affections consist almost exclusively in inflammations where the purulent disintegration has rapidly advanced to the deep parts and involved the bone in a short time. This explains why extensive involvement of the tympanic membrane, granulations in the tympanic cavity, and serious otitic changes of the auricular ossicles are found as early as one or two weeks after the onset of the inflammation. Suppurative

resorption of the ligaments and mucosal folds may soon lead to exfoliation of the auricular ossicles.

The histological picture is that of a violent inflammation and necrosis of the mucous membranes, and in grave cases has all the signs of diphtherial inflammation. Bacterial examination usually shows streptococci in pure culture, sometimes the bacillus diphtheriæ or mixed infections.

The changes occurring in panotitis of the labyrinth correspond to hyperacute diffuse suppuration of the labyrinth. The nerve-endings are entirely destroyed in a short time, and in the further course of the suppuration the entire membranous labyrinth may likewise be destroyed. Empyema of the labyrinth, resulting from these conditions, may be cured spontaneously before the suppurative inflammation has spread to the surrounding bone. Should, however, the suppuration of the labyrinth continue, the inflammatory process will spread to the osseous capsule. There will be all the anatomical signs of complicated diffuse suppuration of the labyrinth (see p. 270), and in some cases sequestra will be expelled. Attention may be called to the possibility of a complicating neuritis occurring in scarlatinal otitis.

The anatomical changes in light cases of scarlatinal otitis or in acute catarrh of the middle ear do not differ from those of the non-scarlatinal form. The same refers to light cases of middle-ear affections occurring in the course of measles.

Frequency and Occurrence.—The frequency of suppurative otitis media in scarlet fever and measles varies, according to the virulence of the prevailing epidemic, between 1 and 33 per cent., as has been statistically demonstrated. The ear affections seldom occur before or during the exanthema; in most cases during the stage of desquamation. In measles they may occur as soon as the fever has run its course. When they occur at an early stage, otitis appears as an enanthematous part manifestation of scarlatina and measles. Light inflammatory changes of the middle ear are very frequent in measles.

One of the most dangerous ear affections occurring in the course of scarlet fever and measles is panotitis. It occurs much more frequently in scarlatina than in measles, and consists in suppurative inflammation of the middle and internal ear. The middle ear and labyrinth are affected simultaneously, or otitis media is followed by suppuration of the internal ear. Panotitis, which usually attacks both auditory canals, occurs more frequently at the climax of scarlet fever than during the period of desquamation or convalescence. It is only exceptionally that both labyrinths are attacked simultaneously, although the interval may not amount to more than a few hours or days. In some measles cases panotitis will not appear before the suppuration of the middle ear has existed for several weeks.

The danger of fulminating, grave inflammations of the ear and of subsequent panotitis is especially great in scarlatinal dipthheria and measles complicated by pneumonia.

Symptoms.—The initial symptoms of scarlatinal and morbillous otitis are those of a severe acute inflammation of the middle ear, consisting in severe headache, considerable dysacusis setting in without any warning, high fever, insomnia, and unrest.

Otoscopic examination (see Fig. 83, 1–5) in the first stages of the inflammation reveals pronounced swelling, bulging, and deep reddening of the tympanic membrane. The swelling is often flesh-like, and the membrane has completely lost its membranous character. Purulent disintegration soon follows. Perforation may occur as early as a few hours after onset of the inflammatory manifestations, and the rapidly spreading suppurative disintegration of tissue often causes large defects of the tympanic membrane inside of a few hours. In spite of careful treatment the secretion will become rapidly fetid, owing to repeated retention or to spreading of the inflammation to the bone. After the suppuration has lasted a few weeks, there will be abundant granulations in the middle ear, and the secretion is mixed with blood from the granulations which are very vulnerable.

The labyrinth symptoms usually set in after perforation of the tympanic membrane, and only exceptionally in the early stages of the otitis. They consist in sudden and considerable impairment of hearing, or deafness, violent labyrinth vertigo, with spontaneous nystagmus, equilibrial disturbances, and vomiting.

Diagnosis.—Taking the above symptoms into consideration, there will be no difficulty in making a diagnosis of otitis media or panotitis. Timely otoscopic examination is of the greatest importance, so that the initial stages of the inflammation may not be overlooked. Accordingly, this is indicated in all cases of scarlatina and measles, even in the absence of any special otitic symptoms. Another point of importance is to continue taking the temperature regularly in acute infectious diseases, even after the fever has run its course. This will guard against overlooking the initial stages of a late otitis in infants or debilitated children.

There is no doubt that in a considerable number of cases the development of suppurative otitis and the spreading of the purulent inflammation to the endocranium or labyrinth is favored by the fact that the initial stages of scarlatinal otitis have been overlooked or have not been treated with the necessary care and energy.

Course and Prognosis.—Scarlatinal and morbillous otitis are rightly dreaded on account of the violence of the subjective and objective pathological manifestations, the rapid disintegration of tissue, the frequency of permanent changes of the middle ear, and the danger of

early spreading to the mastoid. In old neglected cases there is the danger of pus descending toward the submaxillary bone, with inflammation of the articulation and subsequent ankylosis.

The prognosis is not unfavorable, provided correct treatment is instituted at the right time. Cures have often been effected under conservative treatment, but permanent changes of the tympanic membrane and the middle-ear spaces (cicatrization, adhesions, synechiæ, persistent perforations), with consequent hearing disturbances which are usually of a medium degree and permanent, will occur more frequently than in true otitis media. Otitis developing in the course of measles practically resembles scarlatinal otitis, but, as a rule, does not run so severe a course.

Scarlatinal and morbillous otitis tends to chronicity, nearly 25 per cent. of all chronic otorrhœas being traceable to the chronic degeneration of the former. It is for this, if for no other reason, that early and energetic treatment of middle-ear suppuration is indicated.

The course of panotitis is unfavorable in nearly all cases. Unless the first attack of labyrinthitis has led to complete destruction of the hearing ability, total deafness will usually set in a few hours or days afterward. Permanent circumscribed suppuration of the labyrinth is very rare in panotitis.

When recovery has taken place, there will be complete deafness in most cases. Some parts of the cochlea may be spared and some hearing remnants consequently preserved. Such remnants may continue to persist, but they may also gradually diminish and end in deafness, owing to secondary atrophy of Corti's organ and the cochlear nerve. Deafness will then persist and soon lead to deaf-mutism in young children. There is less danger of the labyrinth suppuration spreading to the endocranium. Death from purulent meningitis due to scarlatinal panotitis occurs very rarely.

Treatment.—The principal point to be observed is to institute early and effective drainage of the inflammatory focus of the middle ear. Fatal spreading of the process can be best prevented by early paracentesis and attentive treatment, care being taken that there is always free evacuation of the secretion. In the further undisturbed course, the treatment of scarlatinal and morbillous otitis does not differ from that of the ordinary forms (see p. 156). The same refers to the indications for operative interference if in the course of the affection the mastoid should become involved, or there be signs of any extra- or endocranial complication. In labyrinth manifestations with consequent danger of retention or stagnation of pus in the middle ear, antrotomy is indicated, even if there be no clinical mastoid symptoms. Operation may become necessary at a later stage when the middle-ear suppuration has become chronic and exhibits surgical symptoms (see p. 202).

In panotitis an operation on the labyrinth can only exceptionally be considered in cases where there is danger of the suppuration spreading to the endocranium, thus indicating the presence of a complicated labyrinth suppuration (see p. 270). The details of conservative, symptomatic treatment are described in the chapter on Suppuration of the Labyrinth (p. 265).

2. AFFECTIONS OF THE EAR IN DIPHTHERIA

Etiology. Occurrence.—Diphtheritic inflammation of the external auditory canal does not occur often. It may have been conveyed there from a similar affection of the middle ear, but may also occur with a healthy state of the middle ear. Diphtheritic inflammations of the external auditory canal are usually deep-seated in the anatomical sense, caused by pyocyaneus infection or severe acute or chronic streptococcus infection of the middle ear.

Otitis media occurs less often in the florid stage of diphtheria, more frequently after the acute manifestations and the fever have run their course.

True diphtheria is usually caused by simple spreading of the inflammatory process from the nasopharyngeal space to the ear, and only exceptionally by purely diphtheritic infection by way of the tube. Primary diphtheritic infection was first observed by Burckhardt-Merian.

Symptoms and Course.—It is a noteworthy fact that in infants and young children otitis media occurring in the course of diphtheria may work serious destruction in the tympanic cavity without perforating the tympanic membrane. Genuine diphtheritic membranes are often formed in the middle ear. Extensive necrosis of the osseous parts of the auditory canal (auricular ossicles, mastoid process, temporal bone, and labyrinth) and endocranial complications may develop in grave cases. Spreading of the suppurative inflammation to the labyrinth will cause total deafness and produce all the other symptoms of panotitis.

Otherwise there is no characteristic difference between the diphtheritic and the genuine forms of otitis media.

The diagnosis is not difficult when the picture of otitis media is fully developed and there is diphtheritic inflammation of the external auditory duct. The difficulty is greater when the inflammatory manifestations of the tympanic membrane are but slightly developed and the inflammatory process extends toward the deeper parts. However, careful and repeated otoscopic examination, functional tests, and regular control of the temperature will lead to the correct diagnosis. The latter should be continued in all cases of diphtheria, as in scarlatina and measles, even after the fever has run its course. Should there be a fresh acces-

sion of temperature, the possibility of an inflammatory process of the middle ear should be considered, even in the absence of any particular otitic symptom.

Bacterial examination will decide the question whether the otitis media has been caused by the diphtheria bacillus or by other bacteria, most frequently by streptococcus in the post-diphtheritic stages. This examination is best carried out with the secretion obtained by paracentesis.

Treatment.—Injection of serum is the principal treatment aside from the methods of treatment described in previous chapters. Instillation of lime water has a beneficial effect in cases of diphtheritic inflammation of the auditory canal with formation of membranes. If the external auditory canal is involved, as well as in cases of noma, the inflammation can often not be prevented from terminating in a high degree of stenosis or atresia of the auditory meatus, even with careful local treatment.

The prognosis is not unfavorable, provided the local and general treatment have been instituted in time. Ever since the introduction of serum treatment the frequency of suppurative otitis media seems to have diminished and the course of the affection to have become milder. The danger of panotitis is considerably less in diphtheria than in scarlatina.

3. AFFECTIONS OF THE EAR IN EPIDEMIC PAROTITIS (MUMPS)

Anatomy. Occurrence.—In epidemic parotitis a complication with neurolabyrinthitis is to be feared. The inflammation may be confined to the vestibular or cochlear part of the labyrinth and eighth nerve, but in some cases it attacks the entire eighth and may even spread to the facial nerve, so that the entire nerve bundle of the internal auditory canal will be involved.

It may be assumed that in some of the cases there are acute inflammatory changes of the serous type which are capable of involution. In other cases there is an infectious inflammation which impairs or destroys the physiological function of the internal ear, either directly or owing to secondary degenerative processes of the labyrinth or eighth nerve. The affection is usually unilateral.

The otoscopic examination shows that the tympanic membrane is either normal or but slightly changed. The middle ear is intact except for moderate catarrhal changes. The labyrinth manifestations correspond to the symptom-complex of serous labyrinthitis (p. 264) or circumscribed suppuration of the labyrinth (see p. 265), and only exceptionally to that of diffuse suppuration of the labyrinth.

Course and Prognosis.—The course of otitic affections in parotitis varies greatly. In a large number of cases there may be a complete

cure, with restoration of the physiological function of the labyrinth, even where the hearing ability had been considerably reduced or eliminated at the climax of the inflammation. These are the cases in which parotid labyrinthitis occurs and continues under the picture of serous labyrinthitis. The static labyrinth completely regains its reflex excitability in many cases. Even where the inflammation had involved the entire labyrinth and permanent deafness had set in, owing to circumscribed suppuration of the cochlea, cures have been observed with perfect functional restoration of the static labyrinth.

Mauthner, a member of my department, communicated a case in which the history showed that parotid labyrinthitis (bilateral in this case) had originally attacked the acoustic and static labyrinths, there having been deafness, vertigo, equilibrial disturbances, and vomiting. The patient was examined one year after the cure of parotitis, showing deafness of the right ear, but perfectly normal reflex excitability of the right static labyrinth.

Many cases of parotid labyrinthitis are cured with permanent hardness of hearing. It may also be assumed that diminished excitability of the static labyrinth may persist. The paralysis of the facial nerve is usually completely cured.

The treatment is purely symptomatic, consisting in rest in bed and application of ice-bags to the head. In violent paroxysms of vertigo the room should be darkened, and galvanization of the auditory nerve instituted to produce galvanic nystagmus in the opposite direction to the pathological nystagmus. In cases of persistent, protracted vertigo, narcotic remedies should be considered, either internally or subcutaneously. Rest in bed should, of course, be continued until the nystagmus is under control, even after cessation of the vertigo attacks. After parotitis and the irritative manifestations have run their course, treatment should consist in application of hot air, sudorific measures, and galvanization of the eighth nerve. The latter treatment should be continued for several weeks two or three times a week, with the anode to the ear and a current of 4–6 ma., each application to last from five to ten minutes. I have not seen any results following the injection of pilocarpine or fibrolysin.

4. EAR AFFECTIONS IN TYPHOID FEVER

Anatomy. Occurrence. Course.—Catarrhal affections of the middle ear are of frequent occurrence in the course of typhoid fever. They are usually exudative with accumulation of a yellow mucous secretion. The affection is often combined with an affection of the labyrinth.

Suppurative otitis media with an inflammatory course is but rarely observed in typhoid fever. In these cases suppurative disintegration

of the tympanic membrane occurs at a rapid rate, and there is also the danger of the inflammation spreading early to the bone. Furthermore, these cases are threatened with panotitis, which means extension of the suppurative otitis media to the labyrinth and endocranial complications.

As was mentioned above, the labyrinth affections occurring in the course of typhoid correspond to the type of serous labyrinthitis. Circumscribed or diffuse suppuration of the labyrinth is rarer. Like the rest of typhoid otitic affections, labyrinthitis is more frequently observed unilaterally than bilaterally.

The anatomical changes of the internal ear seem to have an elective preference for the eighth nerve. It is a question whether there are exudative and inflammatory processes going on in the acute stage, as is the case in typhoid middle-ear affections.

Investigations made by Manasse and Sporleder have shown that typhoid deafness is due to an inflammatory process of the auditory nerve or within the labyrinth. In one case of typhoid deafness Manasse demonstrated chronic inflammatory changes of the membranous labyrinth, consisting in new-formation of connective tissue, degenerative atrophy of the neuro-epithelium and the nerve ganglia, and in changes of the osseous capsule of the labyrinth (pathological osseous foci).

Hartmann has called attention to the frequency of otitic affections, notably of catarrh and suppurative inflammation of the middle ear, in exanthematous typhoid. In the latter affection the typhoid exanthema occurs exceptionally at the concha or external auditory meatus, and is usually associated with hemorrhagic vesiculation of the external auditory meatus or tympanic membrane.

Otoscopic Findings and Symptoms.—In cases of exudative catarrh of the middle ear the tympanic membrane is saturated with a yellow secretion (Plate VIII), the line of the manubrium is narrow, and the tympanic membrane is either normal or moderately retracted. If only part of the middle-ear spaces is filled with exudate, the characteristic level line (Politzer) can be observed. Fulminating hemorrhagic inflammation of the middle ear which occurs in the course of typhoid is characterized, like influenza otitis, by the formation of vesicles containing a serous, hemorrhagic, or purulent fluid in the external auditory meatus and tympanic membrane, which is followed later by evacuation of hemorrhagic pus.

In uncomplicated cases the functional test shows a reduction of the hearing acuity and all the signs of obstructed sound-conduction.

The labyrinth symptoms consist in hardness of hearing, subjective noises, paroxysms of vertigo, and equilibrial disturbances. The nervous character of the subjective noises can be definitely established in these cases by means of the galvanic current. If the anode is applied to the

ear (tragus or mastoid) and the cathode at an indifferent place, the subjective noises will be considerably reduced on closing the current, or they may entirely disappear while the current passes through the body. When the cathode is applied to the ear after the current has been reversed, there will be an increase of the subjective noises on closing the current. The intensity of the paroxysms of vertigo and equilibrial disturbances is slight, there being rather a continuous sensation of vertigo without any distinct character of rotation, unstability, etc.

Treatment.—The catarrhal or inflammatory changes of the nose and nasopharyngeal space claim first consideration. If there is spontaneous improvement of the hearing acuity within a few days, indicating resorption of the exudate, no local treatment whatever is needed. Should the accumulation of secretion in the middle ear continue, air may be inflated into the nose after the inflammatory process is over. If, in spite of this treatment, the exudate is not arrested, it should be evacuated by paracentesis (see p. 151).

The treatment of acute typhoid otitis is described on p. 165.

When the labyrinth is affected, the disagreeable tinnitus and the sensation of vertigo may be reduced or temporarily arrested. Correct lateral position of the patient, opposite to the direction of the nystagmus, is a favorable measure. In the presence of nausea, the administration of food requires the greatest care, and none but cool and liquid food should be given in small quantities at a time.

There should be no local treatment in typhoid affections of the labyrinth and eighth nerve. It is questionable whether drainage of the cochlea can be effected and total deafness prevented, in cases of diffuse suppuration of the labyrinth, by opening the lateral semicircular canal and early incision of the pus focus in the static part of the labyrinth.

Prognosis.—The exudative middle-ear catarrhs in typhoid fever are prognostically favorable. Hearing acuity usually returns to normal, but in some cases a moderate degree of dysacusis remains.

Suppurative typhoid otitis does not greatly differ prognostically from morbillous otitis. As in the latter, the danger of panotitis is smaller than in scarlet fever.

It cannot be denied that typhoid otitis has a tendency to chronicity,—a reason why there should be careful treatment in the initial stages of the inflammation. Neglect of these dangerous ulcerations is attended with evil consequences.

Serous labyrinthitis occurring in the course of typhoid may be cured with complete restoration of the labyrinthal function; in circumscribed suppuration of the labyrinth a considerable reduction of the hearing acuity will persist, and diffuse suppuration of the labyrinth will lead to deafness and later to deaf-mutism.

5. EAR AFFECTIONS IN THE COURSE OF INFLUENZA (LA GRIPPE)

Anatomy and Occurrence.—Frequency and intensity of ear affections in influenza vary and may attain a very high degree of virulence in epidemics. The lightest cases, which are of very frequent occurrence, are characterized by transition from simple catarrh to inflammation. Myringitis hæmorrhagica and acute exudative catarrh of the middle ear are not of rare occurrence.

Otitis hæmorrhagica, or briefly designated as influenza otitis, is caused either by the influenza bacillus alone or by a mixed infection (influenza bacillus, streptococcus pyogenes, streptococcus mucosus). Anatomically, influenza otitis is characterized by hemorrhagic exudates into the middle-ear spaces, with abundant secretion of fibrin. The inflammation often spreads to the mastoid in a remarkably short time. Simultaneous involvement of the labyrinth (panotitis) is very rare in influenza otitis. Among the endocranial complications, otogenic influenza meningitis is most dreaded, as it usually runs a hyperacute, fatal course.

Infectious neuritis of the eighth nerve, often combined with neuritis of the trigeminus, abducens, or facial nerves, may occur in the course of influenza, and pronounced nerve hearing disorders after influenza (neurolabyrinthitis) have been reported by various authors.

Symptoms.—In simple catarrh or light inflammation of the middle ear, the tympanic membrane is grayish red, with radial vascular injection and no bulging. Hearing acuity is moderately reduced, the temperature is normal, and the pains are unimportant. In influenza myringitis, blood vesicles will occur in the external auditory canal and tympanic membrane. The external wall of the blood-vessels consists of the epidermal epithelium of the skin or the epidermal layer of the tympanic membrane. The latter may be completely hidden by blood vesicles. In other cases punctiform hemorrhages will occur in the external auditory canal and tympanic membrane.

The onset of typical influenza otitis is characterized by sudden intense earache, considerable dysacusis (3½–6½ feet C.), and high fever.

The otoscopic findings in typical influenza otitis are characterized by blood vesicles in the external auditory canal and tympanic membrane. In the further course of the affection there will be accumulation of a sero-hemorrhagic, and later purulent hemorrhagic, exudate in the tympanic cavity, and finally suppurative decomposition and perforation of the tympanic membrane unless an outlet has been provided for the pus by paracentesis.

The functional test shows severe obstruction to sound-conduction in uncomplicated cases.

After the pus secretion from the external auditory meatus has once commenced, the symptoms of uncomplicated cases do not differ from those of true acute suppuration of the middle ear (see p. 152).

When the labyrinth has become involved, there will be the typical symptom-complex of reduced upper sound-limit, hardness of hearing, spontaneous nystagmus, rotatory vertigo, equilibrial disturbances, and, in very high degrees of development, total deafness and vomiting.

The catarrhal affections as well as myringitis hæmorrhagica admit of a thoroughly favorable prognosis. In the latter affection the vesicles are usually emptied spontaneously after a short existence, the contents of the small vessels being gradually resorbed. Traces of tympanic hemorrhages may remain visible at the tympanic membrane for a long time in the shape of dark red-brown spots. In the end there is complete resorption of the coagula. Pigmentation of the tympanic membrane after hemorrhages is one of the greatest rarities.

Influenza otitis has a tendency to spread to the epitympanum and antrum. Owing to the rapid extension of the inflammation an empyema of the mastoid may develop in a short time. Retention of the secretion in the tympanic cavity will increase the danger of the inflammation spreading to the mastoid, especially to pneumatic ones, where large abscesses will rapidly develop through purulent disintegration of the osseous septa. The external cortical layer will be perforated and a subperiosteal mastoid abscess will be the result. When the medial cortical layer has been perforated, the consequence may be a perisinous abscess.

The treatment of otitis has been discussed in the corresponding chapters on pp. 138, 150, 156. It should be most particularly emphasized that in influenza otitis nothing but timely energetic measures can promote an uncomplicated favorable course and the prevention of complications. It is advisable not to wait for spontaneous perforation, but to effect an early relief of the middle-ear spaces by paracentesis. The principal task of the further treatment consists in maintaining a free outlet of the purulent secretion of the middle ear during the entire period of inflammation.

6. MENINGITIC (MENINGOGENIC) LABYRINTHITIS (MENINGIC DEAFNESS)

Anatomy and Occurrence.—Cerebrospinal meningitis involves considerable danger to the labyrinth.

The meningitic purulent exudate penetrates into the internal auditory canal, and the suppurative inflammation spreads to the interior of the labyrinth over the dural lining of the internal auditory canal and alongside the nerve canals, or by way of metastasis. The result is suppurative peri- and endolabyrinthitis with total destruction of all laby-

rinthal nerve terminations. Histological examination has shown that in the early stages of purulent labyrinthitis the entire neuro-epithelium is destroyed within a few hours. A cure takes place under formation of cicatricial connective-tissue layers in the labyrinth. In some cases there will be obliteration of the labyrinth by connective-tissue formation, which may be followed by osseous obliteration of the labyrinth spaces. Meningitic suppuration of the labyrinth corresponds almost without exception to the diffuse type. Functionally efficient places of the neuro-epithelium, especially in the region of Corti's organ, with clinically demonstrable hearing remnants, are practically never preserved.

Meningitic labyrinthitis usually sets in at the climax of cerebrospinal meningitis at a time when the attention of the family is deflected from the otitic complaints by the grave general symptoms and the onset of deafness is hidden by the disturbed consciousness of the patient. This explains why meningitic labyrinthitis often remains unrecognized until the meningitis has run its course, and the affection of the ear remains unnoticed until at the time of convalescence the condition of deafness becomes apparent.

The causative factor in a large number of cases of acquired deafness is meningitic suppuration of the labyrinth. The affection attacks almost without exception both labyrinths, either simultaneously or with a short intermission. The middle ear either remains perfectly intact or undergoes only catarrhal or light inflammatory changes. Meningitic panotitis of the scarlet fever or measles type is extremely rare.

The otitic findings show the tympanic membrane to be either normal or but slightly changed by catarrh or acute inflammation; sometimes there is vascular injection along the insertion of the membrane.

The onset of diffuse labyrinthitis is characterized by sudden deafness. There are also violent paroxysms of vertigo, equilibrial disturbances, and vomiting if at first but one labyrinth had been attacked. If the suppuration attacks both labyrinths simultaneously, putting them out of action by inflammation, there will be but one paroxysm of vertigo or none at all. The grave general meningitic symptoms may veil the vertigo and its psychic sequelæ, and the spontaneous nystagmus of the labyrinth may be counteracted by the central nystagmus of the meningitis.

Treatment, Course, and Prognosis.—Treatment is powerless in meningitic labyrinthitis, which in the majority of cases will terminate in total and permanent deafness and later deaf-mutism in young children. Older children, who have been able to speak before the affection and acquired deafness after cerebrospinal meningitis, should be instructed in lip-reading at the earliest possible time, as this will prevent the devel-

opment of dumbness. Hearing remnants persisting immediately after meningitis will be preserved quantitatively unchanged in most cases; in others they will gradually decline and finally disappear. Only the future will show whether it will be possible to prevent the occurrence of total deafness, or at least to preserve some hearing remnants, by early operation on the labyrinth far away from the cochlea, perhaps at the eminence of the external semicircular canal.

If labyrinthitis takes a more chronic course, it is possible for the suppurative inflammation to spread first to the capsule of the labyrinth and then, after fistulous perforation, to the middle ear. In this process the labyrinthal fistulæ occur with great frequency in the region of the windows of the labyrinth and the promontory.

7. AFFECTIONS OF THE EAR IN THE OTHER ACUTE INFECTIOUS DISEASES

Affections of the ear in variola are relatively rare. According to Spira they are usually light and occur only in the grave forms of this affection (catarrh and light inflammations of the middle ear).

In rubeola or infectious or varioloid erythema the eruption may spread to the concha and the external auditory meatus.

In rubeola the retro-auricular lymph-glands are nearly always swollen.

In erysipelas the exanthema may spread to the auricular region and the concha, simulating mastoiditis (see p. 179), while in rare cases acute serous labyrinthitis may develop. Cure takes place with restoration of the normal hearing acuity or slight reduction of the same.

In pertussis the ear is not often involved. Acute myringitis or otitis which may develop in the course of violent paroxysms of coughing have also been observed; they do not differ in their course from the true form. Punctiform hemorrhages of external auditory meatus or tympanic membrane may occur exceptionally.

Rupture of the tympanic membrane may occur in paroxysmal coughing if there was previous atrophy or lowered resistance from cicatrization. The violence of the cough may fling the secretion of the nose and nasopharyngeal tract through the tube into the middle ear, causing infection. In a similar way, acute otitis may occur in the course of catarrhal or croupous pneumonia.

In dysentery of nurslings, myringitis, catarrh of the middle ear, or hemorrhagic otitis is of extremely frequent occurrence (see p. 161). A parotid abscess developing in the course of dysentery may exceptionally perforate into the external auditory canal.

Light or medium otitic disturbances may occur at the climax of acute articular rheumatism. Examination will reveal the presence of neurolabyrinthitis. Graver forms of the latter affection, resulting in

permanent dysacusis or deafness, occur only exceptionally in the course of acute articular rheumatism. The use of salicylic preparations in the latter affection may lead to intoxication of the labyrinth and eighth nerve. Examinations by Witmaack and Blau have shown that salicylic acid as well as quinine and antipyrin has an elective preference for the peripheral auditory ganglion as a point of attack. Otitis media occurring in the course of acute articular rheumatism does not differ from the genuine form of that affection.

Osteomyelitis may exceptionally spread to the temporal bone or middle ear, either by direct continuity or by metastasis.

IV. EAR AFFECTIONS IN CHRONIC INFECTIOUS DISEASES

A. TUBERCULOUS DISEASES OF THE EAR

Etiology (Mode of Infection).—The tuberculous diseases of the ear are caused by Koch's tubercle bacillus. The infection takes place by way of the blood or lymph tracts, or exceptionally direct by exposed traumas of the auricular region. The middle ear may also be infected through the tube, especially in cases where the tuberculous virus is present in the nasopharynx, in the oral cavity, or in the larynx.

Otitic tuberculosis is in most cases a secondary affection, the number of primary infections being comparatively small. As to the localization of the latter, the lobule of the ear may be especially mentioned, as well as tuberculous perichondritis, which is of frequent occurrence in childhood, but is often mistaken for other affections.

Many of the cases reported in the literature as primary tuberculosis of the ear are unquestionably secondary, as the clinical demonstration of the primary focus, notably of the lymph-glands and intestines, did not succeed. Brieger and Goercke have pointed out the polymorphous character of the clinical picture of otitic tuberculosis, the cause of which they see in the pathogenic variety of tuberculous ear affections. Tuberculous affections of the middle ear may develop by direct spreading of a tuberculous affection of the nasopharyngeal mucosa by way of the tube or by the formation of isolated tuberculous foci in the middle-ear mucosa. The osseous parts of the middle ear may be infected by tuberculosis simultaneously with the mucous membrane, or the chronic tuberculous suppuration may only spread to the bone after having persisted for some time, the result being tuberculous caries. The tuberculous suppuration of the bone is at times circumscribed and limited to the auricular vessels, the attic wall, and antrum, but not infrequently extends over the entire temporal bone. Tuberculous softening foci develop at the upper wall of the upper auditory canal in a few cases, also at the root of the zygomatic process and the squama of the temporal bone; and these cases present the picture of osteoperiostitis from the first.

1. *Lupus of the Concha. Tuberculosis of the Lobule*

Lupus vulgaris appears at the concha in the same forms as at any other parts of the body. The clinical pictures vary, but their characteristic points consist in the presence of brown-red or brownish lupus nodules the size of a pin-head to a hemp-seed, which are embedded in the skin and do not change on pressure.

Lupus seldom occurs at the concha alone and is much more frequently a part manifestation of a lupous affection of the facial skin. The affected part of the concha is diffusely infiltrated, and the lobule, for which lupus has a predilection, assumes an unshapely, thickened form.

Fig. 121.

Tuberculosis of the lobule of the ear in a child five years old.

Lupus develops at the concha under the same picture as at any other part of the body), namely, as lupus maculosus, exfoliativus, tuberosus, exulcerans, crustosus, erythematodes.

The diagnosis presents no difficulties in typical cases, but combinations of the various forms of lupus may produce polymorphous pathological pictures which are difficult to recognize.

The object of the treatment is to destroy the lupous tissue by applying the caustic stick, chlorate of zinc, the sharp spoon, the Paquelin cautery, or excision. In extensive ulcerations concentrated lactic acid may be tried.

Treatment with Finsen light has been followed by good results; radiotherapy has been equally recommended. Veiel advises application of pyrogallol ointment (0.2–2 per cent.) after cauterization, which is said to conduce to elegant cicatrization.

As a matter of course, general treatment (nourishing diet, etc.) should be strictly attended to, aside from applying local remedies.

Hot-air treatment, after Hollaender, sometimes has a favorable effect. Dreuw's treatment consists in freezing lupus with ethyl chloride, followed by careful cauterization with crude hydrochloric acid. The following have also been recommended: Unguentum viride (Unna), creosote-salicylic plaster and pyrogallol plaster. Zinc-ichthyol ointment is applied in inflammatory irritation; Unna's tuberculin soap for softening lupous fibroma. Where disfiguring rigid scars are in progress of formation, thiosinamine or fibrolysin should be used, as they have occasionally led to very satisfactory cosmetic results (fibrolysin injections, thiosinamine ointment soap, or thiosinamine plaster). Tuberculosis preferably attacks the lobule (Fig. 121), whence it may become diffuse (Fig. 122).

Fig. 122.

Pre-auricular and auricular skin tuberculosis in a six-month-old child.

In these cases tuberculosis seems to be occasioned by a local infection that has occurred by inoculation with tuberculous virus. The principal cause seems to be the puncture of the lobule for ear-rings, since recurring eczema or other irritations of the little canal may cause conditions favoring local infection. Haug found tubercle bacilli and a caseated tuberculous nodule in angioma of the lobule which had existed for 22 years.

Tuberculosis of the concha develops very slowly with late ulceration.

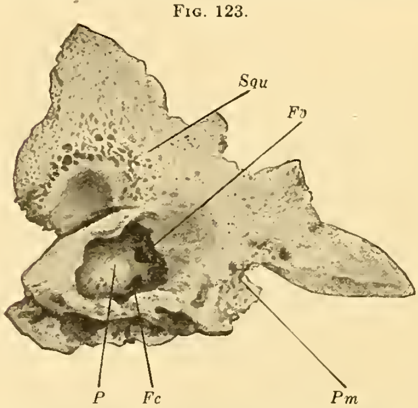
The neighboring glands are usually involved. The prognosis is by no means unfavorable, as the course of the process is usually benign.

2. *Tuberculous Perichondritis*

Tuberculous inflammation of the cartilaginous integument is a rather rare affection. It occurs more frequently in young people than in the old. In most cases there seems to be a local tuberculous inflammation in otherwise healthy individuals.

Symptoms.—Tuberculous perichondritis usually sets in under the picture of painless furunculosis of the external auditory meatus, less often under manifestations of phlegmonous perichondritis. Characteristic symptoms are the painless and afebrile, protracted course, livid discoloration of the skin, gradual extension of the infiltration, and perhaps abscess formation at the base of the concha (descending tuberculous abscess). A fistula is usually formed spontaneously at the medial surface of the concha at the fusion of the auricular cartilage with the concha. Sometimes the pus perforates through one of the congenital osseous fissures or near the lobulus.

Fig. 123.



Tuberculosis of the temporal bone in a child of five years. Carious destruction of the squama (*Squ*), the zygomatic process, and the mastoid (*Pm*). *Fe*, fenestra vestibuli; *Fc*, fenestra cochleæ; *P*, promontory.

Treatment.—Treatment with the Finsen light is useful in cases where no abscess has yet been formed. In the presence of fluctuation or of a fistula, surgical measures have to be instituted under anæsthesia. The abscess region is exposed by an incision of the concha, curetted with the sharp spoon, and tamponaded with ½ per cent. formalin gauze. Lactic acid has also proved very serviceable in cauterization of tuberculous granulations.

After the cure a permanent deformity of the cymba conchæ and the posterior line of insertion will nearly always remain.

3. *Tuberculous Otitis Externa*

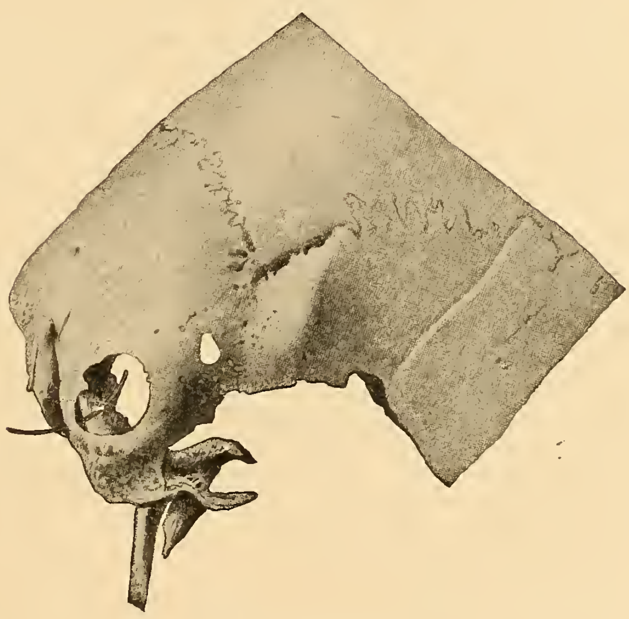
This otitic affection which sometimes occurs in childhood is wrongly named, since there is no question of a tuberculous affection of the sebaceous glands, but always of tuberculous perichondritis of the auricular cartilage. The inflammation sets in under the picture of an auricular furuncle, but painless. The course likewise is painless. At first the infiltration may remain almost unchanged for several weeks; finally, however, the swelling will extend to the cavity of the concha, and sooner

or later there will be fistulous perforation to the base of the ear, a fistula developing at the lower end of the line of insertion of the concha with all the characteristic symptoms of tuberculous fistulæ (thin, livid edges, dilute, weakly fetid, aqueous pus). The treatment consists in free exposure of the infected part of the concha by means of a cutaneous incision in the line of insertion, passing through the fistula. After removal of the ulcerated parts with the sharp spoon, the wound cavity is tamponaded with formalin gauze. When there are signs of a tuberculous inflammation of the external auditory meatus, it is important to consider the persisting scars and the possibility of a permanent stenosis of the auditory canal or deformity of the cavity and base of the concha. Other tuberculous foci which may require local and general treatment should also be looked for.

4. Tuberculous Affections of the Middle Ear

Isolated tuberculous inflammations of the tympanic membrane with formation of fibrous, purulent, disintegrating nodules do not often occur.

Fig. 124.



Extensive caries of the temporal bone with complete destruction of the middle ear and formation of multiple fistulæ.

As a rule, the tuberculous changes of the tympanic membrane are but a part manifestation of tuberculous suppuration of the middle ear.

The history usually gives no precise information on the beginning of the suppuration, as the tuberculous inflammation sets in painlessly and the affection of the ear is often not recognized until there is a puru-

lent secretion flowing out from the auditory meatus. The inflammation is characterized in many cases by a particularly small perforation. Perforations the size of a pin-head or a mere puncture are by no means rare (Fig. 127). There are also cases with multiple perforations (Fig. 127). Tuberculous suppuration with considerable defects of the tympanic membrane nearly always belong to the group of those cases in which an originally non-tuberculous suppuration has been secondarily infected.

Fig. 125.

Tuberculous caries of the right temporal bone of a child, four years old, with complete destruction of the mastoid (*M*). *Ty*, tympanic cavity.

The epidermal layer of the preserved part of the tympanic membrane may be found either intact or ulcerated and granulating. The purulent secretion is remarkably thin and often highly fetid. After the tuberculous suppuration has spread to the bone (Figs. 123–126), all the cranial manifestations of the surgical forms of chronic middle-ear suppuration will of course develop (see p. 191). Cholesteatoma, pyorrhœa of the tube, and granulations of the pharyngeal tube opening may likewise appear.

In uncomplicated cases the functional tests will reveal the signs of obstructed sound-conduction, and in most of them the hearing acuity is considerably impaired. After the affection has spread to the internal ear, there will also be labyrinthal symptoms (slight hearing remnants or deafness, vertigo, equilibrial disturbances, etc.). In extracranial complications subperiosteal abscesses will make a painless appearance and may attain a considerable extension, owing to the pus gradually lifting ever-increasing areas of periosteum from the bone, the connection between them being very loose in tuberculous

Fig. 126.

Tuberculous mastoiditis with complete disintegration of the corticalis and the posterior wall of the auditory meatus. *Mae*, external auditory meatus.

individuals. Thus, I observed in a tuberculous child, eight years old, a perforation of a tuberculous mastoid abscess through the posterior wall of the auditory canal. The integument of the canal, however, remained intact and was bathed in pus, which finally spread, through ossification gaps of the tympanic bone, to the submaxillary region and thence along the medial surface of the submaxillary bone to the base of the oral cavity. Subperiosteal, tuberculous mastoid abscesses may attain an astonishing extension by spreading to the nape of the neck and the occipital and parietal bones. Mixed forms of cholesteatoma and tuberculous suppuration may likewise occur. Preysing has described a case of a tumor-like tuberculosis of the middle ear. Tuberculosis of the mucous membrane of the tympanic cavity with an acute onset has been observed by Brieger, Jansen, and Kuemmel.

Fig. 127.

1 2

Chronic, tuberculous suppuration of the middle ear. 1. Double perforation: defect of Shrapnell's membrane (right) with partial destruction of the lateral attic wall; the second perforation is close below the umbo. 2. Chronic, tuberculous middle-ear suppuration (right); multiple perforation of the pars tensa.

The temperature is usually normal. The general symptoms include ill appearance, anorexia, and anæmia. These signs are also observed in cases where the tuberculous otitic affection is the only complaint and where the internal organs are perfectly free from tuberculosis.

Course.—Tuberculous middle-ear suppuration has a tendency to a chronic course from the onset. Three types may be distinguished: (1) Tuberculous middle-ear suppuration where all the characteristic signs of tuberculous suppuration will persist unchanged; (2) cases which set in under the picture of middle-ear tuberculosis but in their further course correspond to the non-tuberculous forms; (3) cases of apparently or positively non-tuberculous middle-ear suppuration in which the signs of middle-ear tuberculosis suddenly manifest themselves.

Brieger's statement, "There is probably no form of chronic middle-ear suppuration which might not be occasioned by tuberculosis," is perfectly justified, especially in view of the large number of cases of tuberculous middle-ear suppuration in childhood.

Koerner separates the incurable form of tuberculous middle-ear suppuration occurring in the terminal stage of pulmonary tuberculosis from curable tuberculosis of the stationary forms.

Prognosis.—Conservative treatment may effect a complete cure in a small number of tuberculous cases of middle-ear suppuration (see pp. 183 and 401). These include tuberculosis of the mucous membrane with an intact osseous middle ear and an otherwise perfectly healthy organism. In most cases, however, the osseous middle ear is likewise affected. Timely radical operation in these cases may not only cure the initial

tuberculosis of the ear, but also have a favorable influence upon the general condition. But there are also cases in which a perfect formation of epithelium cannot be attained in spite of a favorable healing process without reactions, and ulcerations with granulation will occur from time to time at circumscribed places of the mucous membrane of the tympanic cavity.

Cases where middle-ear tuberculosis is only a part manifestation of a general tuberculous affection of the organism are prognostically unfavorable. These include chiefly the cases which are complicated by glandular and pulmonary tuberculosis as well as those that are associated with multiple caries of the skeleton. Under these circumstances middle-ear tuberculosis continues to spread in the ear with formation of sequesters, and finally caries of the petrous bone will develop (Figs. 123–126), with necrosis of the labyrinth (Fig. 95) and paralysis of the facial nerve. The tuberculous suppuration may extend to the carotid in rare cases, leading to sudden death by carotid hemorrhage owing to erosion of the carotid artery. In most cases death is caused by an endocranial complication (tuberculous meningitis or cerebral tumor). Tuberculous middle-ear suppuration may also occasionally be complicated by an acute suppurative, non-tuberculous endocranial affection.

The general nutritive condition of the patient is impaired by middle-ear tuberculosis in a considerable number of cases. This refers especially to bilateral tuberculous middle-ear suppuration with profuse secretion, where a tuberculous affection of the nasopharyngeal mucosa occurs after some time, which leads almost without exception to a rapid advance of the pathological process in the lungs and to rapid marasmus. But even grave cases of otitic tuberculosis may exceptionally recover, as shown in a case of Brieger, where, in spite of extensive destruction of the mastoid and the presence of tuberculous pachymeningitis, a cure was effected.

Diagnosis.—The clinical diagnosis of tuberculous middle-ear suppuration does not ordinarily meet with any great difficulties. Many cases are characterized by absence of pain and reactions in the beginning of the affection. The late occurrence of perforation or the presence of two or more perforations of the tympanic membrane (Fig. 127) may furnish valuable guiding points for the diagnosis. Persistence of particularly small perforations is suspicious, when the suppuration has been present for a long time. Tuberculin injection may decide the diagnosis in many doubtful cases. A negative reaction to a puncture of 1 mg. tuberculin definitely excludes active tuberculosis. Positive puncture reaction as well as the Pirquet reaction, however, does not admit of a conclusion as to the nature of the otitic affection. Microscopic demonstration of tubercle bacilli in the pus taken from the ear will only suc-

ceed in exceptional cases. Histological examination of the granulations, however, will always admit of a positive diagnosis of tuberculous middle-ear suppuration by the demonstration of characteristic tuberculous giant cells or of tubercle bacilli in the tissue, which stain after Ziehl. The bacteriological examination of a local lymph-gland, which may be removed on the occasion of a radical operation, may at times confirm the diagnosis of tuberculosis of the ear. Ear polypi, however, but rarely show characteristic histological signs of tuberculosis.

Conservative Treatment.—The conservative treatment of tuberculous middle-ear suppuration does not materially differ from the treatment of the non-tuberculous form. The application of strong antiseptics (iodoform, iodol, etc.) should be continued for a long time. Many authors recommend instillation of 30 per cent. lactic acid or 1–2 per cent. trichloracetic acid solutions. Insertion of cotton plugs moistened with balsam of Peru or cauterization with concentrated aqueous picric acid solution, lactic acid, or chromic acid sometimes leads to rapid and considerable diminution of the secretion. Furthermore, irrigations with aqueous formalin or sublamine solutions (1 Gm. of commercial concentrated formalin solution to 1 pint of water, sublamine solution 1 to 1000) and insertions of gauze saturated with formalin, sublimate, or sublamine are to be recommended. In other cases the stagnation and aspiration treatment seems to have a favorable effect. Conservative treatment may lead to complete cure if the tuberculous suppuration has been confined to the mucous membrane of the middle ear or the osseous parts of the middle ear are affected at superficial and easily accessible places, especially if the patients are otherwise healthy and robust.

A valuable adjuvant is the local application of light. A hard-rubber funnel of the greatest admissible amplitude is introduced into the auditory meatus and the patient brought into such a position that the full sunlight may enter the funnel, while the rest of the head is protected against the sun's rays. This may be done in the open air, weather permitting. A metal or glass reflector will serve to conduct the rays into the ear when the proceeding takes place in a room. When the weather is dull or cold, the Auer lamp may be used. The results of illumination are exceedingly favorable, not only in conservative treatment, but also in the treatment after operations, as it leads to considerable reduction of the secretion and rapid formation of epithelium. I have not seen any advantage from employing blue light.

General invigorating treatment is of fundamental importance in all cases of middle-ear tuberculosis. If it is possible by dietary means to effect an increase in body weight, the chances of a cure in tuberculous middle-ear suppuration are favorable. In order to make sure of correct general treatment, it is advisable to place patients in an institute for

tuberculosis, provided there is an ear specialist attached to it who is conversant with the methods of treatment. Otherwise a stay in the country in a warm, dry, and wind-protected district, together with milk treatment and a fattening diet, is often attended with remarkable success.

Indications for Operation.—Surgical interference in tuberculous middle-ear suppuration should be considered if, after one or two weeks' conservative treatment, fetid suppuration should continue and all those manifestations occur which have been mentioned when discussing the surgical forms of chronic middle-ear suppuration. Operation, however, should only be resorted to if the general condition of nutrition is favorable and there is no fever. Where the suppuration has not existed for more than a year and the condition of the patient is satisfactory, antrotomy will be found sufficient; attico-antrotomy is indicated if there be changes in the attic. In all other cases the typical radical operation should be carried out. A successful operation depends upon the question whether all the affected bony parts have been removed and also on the condition of the patient previous to operation. A favorable prognosis may be made for those cases whose weight could be increased before the operation by appropriate diet. The healing process in these cases does not ordinarily differ from that in radical operation in non-tuberculous middle-ear suppuration. Less favorable is the prognosis in cases where the nutritive condition has been impaired through many years of suffering and where an increase in weight before operation does not occur as a result of appropriate diet. In a considerable number of these cases, however, the radical operation exercises a visibly favorable influence upon the nutritive condition, effecting a rapid increase in weight. The healing process is rapid in some cases, while in others it takes from six to eight months before complete recovery. In those few cases where the epidermis is not completely restored, we must content ourselves with the fact that the secretion from the middle-ear spaces is no longer fetid, that it can be satisfactorily drained off, and that in the course of time it assumes a purely mucous character. Unfortunately, this ulceration of epithelialized places from time to time cannot be prevented, as the new-formation of epithelium goes on. But even in these cases the favorable effect of removing the otitic focus upon tuberculous foci in other parts of the body can be recognized as well as the favorable indirect influence of the radical operation upon pulmonary and glandular tuberculosis, should such exist.

Prognostically unfavorable are those cases in which advanced middle-ear tuberculosis is associated with multiple caries of the skeleton or progressive pulmonary tuberculosis. Operation will then be resorted to only in cases of urgent necessity owing to a complicating endocranial

affection or labyrinth suppuration. A cure of the otitic tuberculosis is out of the question in these cases. Greatly debilitated individuals will only slowly recover from the operation. The operative wound is almost of a cadaverous odor during the first few weeks; there is practically no traumatic secretion, merely a sparse, viscid layer. These patients may give the impression as if the skeletal, glandular, and pulmonary tuberculosis had actually become worse by the operation, owing to the general debilitation of the patient. In advanced middle-ear tuberculosis and extension of the affection to the cranial surfaces of the temporal bone and labyrinth, there is also the imminent danger of tuberculous meningitis or cerebral tuberculosis setting in, both of which nearly always run a fatal course.

5. Tuberculous Infections of the Internal Ear

Habermann, Herzog, Kuemmel, and Politzer agree in pointing out the frequent involvement of the labyrinth in tuberculous suppuration of the middle ear.

Tuberculous suppuration of the labyrinth may underlie an involvement of the labyrinth, but cases have also been reported where tuberculous middle-ear suppuration was associated with non-tuberculous suppuration of the labyrinth.

Anatomy.—Tuberculous suppuration of the labyrinth may be confined to some part of the labyrinth for a long time and only become diffuse at a subsequent period. In the early stages of the affection there is a serous exudate in the labyrinthal spaces, swelling of the membranous walls of the labyrinth, and inflammatory infiltration. Simultaneously, or shortly afterward, the sensory cells of the labyrinthal nerve-end places are destroyed. Later changes include accumulation of pus in the labyrinth spaces, secretion of a fibrinous exudate, and the partial obliteration of the labyrinth spaces by connective-tissue proliferation. These changes in the hollow spaces of the labyrinth are accompanied by changes in the osseous capsule and the petrous bone, but the para- and endolabyrinthal inflammatory manifestations do not occur simultaneously, and the time of their appearance differs in individual cases. As a rule, the tuberculous suppuration of the petrous bone precedes that of the membranous labyrinth.

The suppurative destruction of the bone is also associated with sequestration, which may involve all parts of the labyrinth. Small sequesters of the promontory, modiolus, and facial canal may be expelled through the external auditory meatus, while large labyrinthal sequesters usually remain for a long time in their normal topographic position. The suppurative destruction of the bone causes granulation and the sequesters are infiltrated with granulations. In sequestration of the

cochlea or of the entire labyrinth, all the hollow spaces of the latter may be replete with granulations. The suppuration of the labyrinth even spreads to the auditory canal in a few cases, leading to purulent infiltration and in chronic cases to purulent decomposition of the nerve tuft. Labyrinthal fistulæ are of exceedingly frequent occurrence, most of which are of the centrifugal variety (see p. 224), which do not appear until the labyrinth has completely exulcerated by outward perforation of the endolabyrinthal abscess. Sequestration of the labyrinth also leads to purulent infiltration and later to complete disintegration of the facial nerve in certain cases.

Symptoms.—The symptomatology of tuberculous suppuration of the labyrinth does not differ from the non-tuberculous forms except that it has a tendency to chronicity and sets in insidiously similar to tuberculous middle-ear suppuration. This may account for the fact that the intensity of the characteristic labyrinthal symptoms is only slight and some symptoms may be entirely absent.

The impairment of the hearing ability induced by the middle-ear suppuration gradually increases and terminates in total deafness. Sudden deafness does not often occur in tuberculous suppuration of the labyrinth. The vertigo has all the characteristic signs of the labyrinthal variety, but, as a rule, is not very intense and does not occur in abrupt paroxysms, but rather in a chronic form. Labyrinthal disturbances of the equilibrium are always present in diffuse tuberculous suppuration of the labyrinth, in the circumscribed form, however, only when the static labyrinth has been involved. The temperature is usually normal. There is often headache on the affected side.

Paralysis of the facial nerve may occur in the beginning of tuberculous labyrinthitis or during its course, and is occasioned by the fact that a tuberculous paralabyrinthal focus has spread to the facial canal. Cases of extensive caries of the petrous bone, however, have also been reported where the facial nerve was intact.

Course.—Tuberculous suppuration of the labyrinth is characterized by a pronounced chronic course. Spontaneous cure is not impossible if the suppuration is confined to the hollow spaces of the labyrinth and the petrous bone remains normal, especially on the surfaces facing the cranial fossæ. These favorable cases heal with complete destruction of the labyrinthal nerve-end places and partial or complete obliteration of the labyrinth by connective tissue which later changes into osseous substance. In other favorable cases healing occurs after expulsion of a number of small sequesters, often amounting to 10 or 15. Cases where endocranial fistulæ develop take an unfavorable course, as they involve the danger of the tuberculous suppuration spreading to the endocranium.

Diagnosis.—The diagnosis of suppuration of the labyrinth as such does not present any particular difficulties, but its tuberculous character cannot usually be recognized with certainty before operation. The clinical signs of the tuberculous character of the middle-ear suppuration and the expulsion of labyrinth sequesters are aids to diagnosis; the protracted course and relatively slight intensity of the labyrinth symptoms, as well as the permanently normal temperature, likewise favor the assumption of a tuberculous process.

Treatment.—Immediate radical operation in the early stages of labyrinthitis is indicated. If the symptoms are fully developed on admission of the patient, a waiting attitude is in order except in the presence of complications. Rest in bed is prescribed, and the paroxysms are tentatively controlled by the galvanic current. They are relieved and shortened by darkening the room and placing the patient in the most favorable position (see p. 269). These measures are accompanied by extremely careful conservative treatment of the tuberculous suppuration of the middle ear with a view to securing free evacuation of the pus. After the labyrinthal symptoms have subsided, the radical operation or, if indicated, resection of the labyrinth may be proceeded with.

In cases of complicated tuberculous labyrinthitis immediate resection of the labyrinth is indicated, although the result is not expected to be satisfactory. The result of the operation itself is often favorable enough, in spite of extensive resection of the petrous bone, but for weeks or months afterward there is considerable danger of tuberculous meningitis setting in, to which the patient will succumb.

B. SYPHILITIC AFFECTIONS OF THE EAR

1. Acquired Syphilis in Childhood

Acquired syphilitic affections are rare in infancy and childhood. They are nearly always occasioned by extragenital infections by kissing, using eating and drinking utensils of syphilitic individuals, or by secondary traumatic infection. Nurslings have been infected by syphilitic wet-nurses.

This form of syphilis rarely extends to the ear. Here and there syphilitic roseola of the concha, with circumscribed papules, has been observed. Both dry and exuding papules may occur at the external auditory meatus.

The local treatment consists in insertions of gray ointment and painting with iodoform and sublimate. Energetic antisyphilitic treatment promptly effects a cure.

I have never observed condylomata of the external auditory meatus, pustulous syphilides, or gumma in the auricular region of children, nor any inflammatory middle-ear affection which could have been accepted as acquired syphilis free from objection.

One case of chronic middle-ear suppuration, however, deserves mention. An extragenital, syphilitic infection occurred in earliest infancy. The child was treated and acquired at the age of five a middle-ear suppuration which became chronic in spite of treatment. After institution of antisyphilitic treatment the middle-ear suppuration was promptly cured.

Syphilitic affections of the labyrinth or eighth nerve in acquired, extragenital syphilis in childhood have not come within the range of my observations, but it may be assumed that the course and prognosis of such affections are perfectly identical with syphilitic affections of the internal ear in adults. For this reason a detailed discussion of these affections may be dispensed with at this place, and I refer, in regard to treatment, to my treatise on syphilitic affections of the ear in the "Handbook of Sexual Diseases."

2. Hereditary Syphilitic Diseases of the Middle Ear

Hereditary syphilitic affections of the middle ear are far less prominent than similar manifestations of the internal ear in regard to frequency and clinical importance.

Asai concluded, from his anatomical investigations of the ears of children with hereditary syphilis, that the latter does not predispose the fœtus or new-born to middle-ear inflammations. The frequency and nature of otitis media are the same whether the ear is or is not infected with hereditary syphilis.

Among the middle-ear infections, suppurations with a grave course are most frequently observed, but they are only indirectly caused by hereditary syphilis. They usually occur in nurslings who present the typical nasopharyngeal changes of hereditary syphilis and are caused by the latter. The unfavorable prognosis of these cases is occasioned by the inferior nutritive condition of all nurslings with grave general manifestations of congenital syphilis.

Children with congenital syphilis sometimes suffer from exudative middle-ear catarrh. In some cases this does not differ from the ordinary, non-syphilitic, exudative catarrh, and is occasioned by nasopharyngeal changes. Occasionally, however, such an exudative catarrh partakes of a specific nature and appears to be a part manifestation of a syphilitic affection of the middle ear and labyrinth. This is evidenced by the fact that after resorption or evacuation of the exudate there is only slight or no improvement of the hearing ability, and the middle-ear symptoms are gradually replaced by those of an affection of the internal ear.

These apparently catarrhal affections develop into affections of the labyrinth or ear which clinically correspond to the type of otosclerosis.

Note.—The affections of the external ear in hereditary syphilis perfectly correspond to those of the integument which have been acquired *intra uterum.* Therefore, they do not differ from acquired syphilitic affections of those regions.

3. Affections of the Internal Ear in Hereditary Syphilis

Owing to their relative frequency in infancy, their grave course, and peculiar findings, affections of the inner ear and eighth nerve in hereditary syphilis command considerable clinical interest. Authors differ as to frequency. According to my own observations, about 6 per cent. of infants with hereditary syphilis suffer from an affection of the sound-perceiving apparatus, with a high degree of dysacusis, deafness, or considerable labyrinthal vertigo. Slight lesions of the internal ear in congenital syphilis which are associated with tinnitus aurium with normal or slightly impaired hearing acuity, with slight labyrinthal vertigo and equilibrial disturbances, are far more frequent.

Anatomy.—The anatomical foundation of affections of the internal ear in hereditary syphilis is not yet quite understood. There is no doubt that there are several anatomical types. The first place is occupied by degenerative neuritis of the eighth nerve, next follow processes in the acoustic labyrinth itself which are accompanied by exudation and coagulation in the spaces of the acoustic labyrinth or by labyrinthal hemorrhages and rapidly lead to degeneration of the neuro-epithelium and the nerve branches. Another type consists in congenital changes of the osseous capsule of the labyrinth, which are probably the result of an inflammatory syphilitic affection of the bones which the fœtus has undergone in intra-uterine life. The histological changes seem to be identical with those found in other bones. Meningitic changes in the new-born which, according to Mayer, may cause interstitial inflammation of the eighth nerve and subsequent development of an otitic affection are likewise due to intra-uterine, syphilitic inflammation. The case observed by Haike likewise belongs to this group. Asai has made histological examinations of the ears of fourteen cases with hereditary syphilis, ranging from a seven-months fœtus to two-months-old infants. The cadaver of a three-weeks-old infant with hereditary syphilis showed signs of a recent pachymeningitis and labyrinthitis, but he could not find in a single case any syphilitic changes of the blood-vessels or pathological hemorrhages of the ear.

Etiology and Occurrence.—It is not yet definitely decided whether luetic endarteritis in childhood is an etiological factor in the occurrence of affections of the labyrinth and eighth nerve. Nor is the cause of the location in the internal ear ascertained. The otitic affection occurs sometimes as a sequel to severe traumas or infectious diseases, this

factor, however, being only an indirect cause of affections of the ear and eye in children with hereditary syphilis. Thus, the majority of cases of deafness and blindness are etiologically traceable to hereditary syphilis.

The most severe forms of hereditary syphilis seem to be acquired during intra-uterine life, the new-born in these cases showing all the signs of congenital deafness and non-excitability of the static labyrinth. A more or less light degree of impaired hearing in young children with hereditary syphilis is of frequent occurrence during the first few years of life. At first it remains stationary, and does not usually undergo any exacerbation until puberty or at the age of eighteen to twenty, when it may degenerate into pronounced dysacusis or deafness.

A third type of syphilitic affection of the labyrinth consists in slightly impaired hearing with tinnitus aurium, or merely in tinnitus without any appreciable impairment of the hearing faculty. Gradual deterioration of the latter may occur, but many of these cases admit of a good prognosis in regard to hearing acuity. These three otitic affections do not often occur singly, they being usually associated with Hutchinson's teeth and interstitial keratitis, or either. The manifestations of the internal ear sometimes occur in an apoplectiform way without any prodromal signs, or at least only with such prodromal signs as escape the attention of the parents, and lead in a few days or weeks to total bilateral deafness, followed by deaf-mutism in earliest childhood. This type is nearly always associated with interstitial (parenchymatous) keratitis, the latter usually setting in after the otitic affection has run its course. In regard to other points, the extent of the labyrinthal affections varies. In most cases the cochlearis and vestibularis are affected, so that the symptoms of both are present. The vestibular disturbances, however, are often slight, and the reflex excitability of the static labyrinth is preserved, while the affection in the region of the cochlearis attains to a high degree and terminates in complete elimination of physiological function, which means deafness.

Next come those cases where deafness occurs together with loss of excitability of the static labyrinth, the entire internal ear having lost its function. As mentioned above, isolated slight cochlear and vestibular affections are very frequent in hereditary syphilis.

The characteristic changes of the reflex excitability of the vestibularis will be referred to presently.

Otoscopic Findings and Functional Test.—As a rule the tympanic membrane is perfectly normal, in other cases osmotic, and sometimes exhibits a reddish glistening in the region of the promontory. Should the middle ear be simultaneously affected, there will, of course, be many catarrhal or purulent inflammatory changes of the tympanic membrane and cavity.

The functional test presents the picture of an affection of the sound-perceiving apparatus, but there are two very characteristic peculiarities which in doubtful cases may lead to the diagnosis of hereditary syphilis. All investigators have found a relatively great reduction of the cranial conduction of the cochlear part. I have also found this to be the case in all the cases I have observed; even in slight impairment of hearing and slight affection of the internal ear, the sound-perception through the cranial bones is considerably shortened in hereditary (and often also in acquired) syphilis.

As to the static labyrinth, abnormalities in the reflex excitability of the semicircular canals are of frequent occurrence in hereditary syphilis. The first place is occupied by positive compression or aspiration nystagmus (positive fistula symptom without a fistula). In affections of the labyrinth due to hereditary syphilis in which there never was any suppurative affection of the ear, nor any manifestations of the static labyrinth, the fistula symptom without the presence of a fistula may be observed in some cases. This is evidently due to changes of the excitable parts of the nerve terminations of the static labyrinth. In normal individuals compression or aspiration of the air in the external auditory meatus only influences the function of the cochlea (Gellé's test), while compression and aspiration in the normal static labyrinth has no physiological effect. In labyrinthal affections due to hereditary syphilis a positive fistula symptom can often be elicited; it may occur periodically either in compression or aspiration (see p. 237).

Diagnosis.—The diagnosis is not beset with any difficulties. The other signs of hereditary syphilis, notably the previous parenchymatous keratitis and the changes of the upper inner incisors, lead to the correct diagnosis. The diagnosis is verified by a positive Wassermann test, which is peculiarly suitable for the diagnosis of congenital syphilis and is positive in the majority of all cases.

Characteristic signs are the occurrence of the affection in childhood, the rapid development of the pathological manifestations, and the above symptoms elicited by the functional test, consisting in unusual reduction of the cranial conduction and fistula symptoms without a fistula. Doubtful cases may be elucidated by the family history and, if possible, examination of the parents.

In establishing the differential diagnosis, there is first the so-called idiopathic atrophy of the auditory nerve to be considered. This affection is very rare, and many cases which were formerly interpreted as such would at the present time show a positive Wassermann reaction and be recognized as hereditary syphilis.

The next affection to be considered is hereditary non-syphilitic hardness of hearing which is usually of a light degree. I have observed and

reported a case of this kind (see p. 234). With aid of the Wassermann test the correct diagnosis may be arrived at at once. Besides, interstitial keratitis and the changes of the incisors are, of course, absent in non-syphilitic cases.

Atypical cases of otosclerosis should be considered in the differential diagnosis. The latter can be made from a negative Wassermann, from the insidious onset and course of the affection, which will not exhibit a pronounced degree until puberty, and from the fact that in otosclerosis the involvement of the static labyrinth nearly always occurs at a late stage. The family history should also be considered, as it may show the tendency to hereditary otitic affections (progressive hardness of hearing or deafness, but no deaf-mutism in preceding generations, nor syphilis).

Treatment.—The general treatment claims principal attention. Immediate salvarsan treatment is indicated in all cases of grave affections of the labyrinth and auditory nerve. In light, fresh cases salvarsan is best preceded by mercury treatment.

Should any exacerbations occur during or after salvarsan treatment, continuation of the antisyphilitic treatment is urgently recommended. My position on this question is still to apply first inunction or sublimate injections and follow this up by salvarsan, while other authors recommend the immediate application and repetition of salvarsan injections. However, the selection of the remedies (mercury and iodide) and their dosage have to be adapted to the peculiarities of each case, the constitution of the child, etc. As to the details of the various methods, the text-book on syphilography should be consulted. Galvanic treatment of the eighth nerve seems to arrest the advance of degenerative changes of the nerve. Children should have 2–4 ma. with both electrodes applied to the tragi three times weekly for four to six weeks, each application to last five minutes. I have not seen any favorable results from pilocarpine injections in otitic affections of heredo-syphilitic origin.

The prognosis is not favorable in the majority of cases. In a number of cases the affection of the labyrinth may remain stationary, or even be cured, with good hearing function. A perfectly normal acuity with sensitive function, however, has never returned in any of the cases. In most of them a gradual or sudden deterioration will set in, terminating in unilateral or bilateral dysacusis or total deafness. The stormier the onset of the labyrinthal manifestations the more unfavorable is the prognosis. An improvement of high degrees of dysacusis or total deafness will not occur by local or general treatment except in the rarest of cases, and then only in fresh ones.

XVII. IMPAIRED HEARING DURING SCHOOL LIFE. THE SCHOOL OTOLOGIST

The strides made in the education of the deaf-mute have caused attention to be paid to this question in the schools. Bezold has shown the influence of impaired hearing on the mental development of school children on the basis of comprehensive statistics. He has shown that school instruction is attended with far inferior results in children whose hearing faculty is impaired than in normal ones, unless due regard is paid to the affliction.

In German and Austrian schools a rule was established many years ago for hard-hearing children to sit on the front bench, so as to facilitate their participation in the lessons. If the affection is of such a pronounced degree that in spite of this rule a child is unable to follow the lessons, an arrangement can be made in classes with a small number of pupils for the former to sit in immediate proximity to the teacher. In the public schools of large cities this arrangement can, of course, not be carried out without disturbing the lessons.

Hartmann was the first to point out that the establishment of special classes for pupils very hard of hearing is not only advisable, but urgently necessary.

When, in Berlin, classes for children with impaired hearing were established, after the school physician had directed attention to the subject, the teachers were requested to send hard-hearing pupils, who could not follow the lessons, forward for examination. In a district containing 23,000 pupils, 45 were selected for examination, but only 30 were admitted. Some of the remaining 15 could be improved by appropriate treatment; in the rest of the cases parents refused to have their children sent to special classes. The 30 children were instructed by two teachers in two sections.

From this experience it may be assumed that, in towns of 150,000–200,000 inhabitants, special schools should be established for children with impaired hearing.

Children commanding a hearing distance of 3 feet 4 inches to 1 foot 8 inches for whispered language can still follow the lessons in public schools, but most of those whose hearing distance is reduced further should be placed in special classes for hard-hearing pupils or receive private lessons.

Hartmann further points out that those acoustically afflicted should be examined by a competent specialist and that, if necessary, treatment

of the underlying affection should be taken in hand. The pupils assigned to a special class should be seated near the teacher, and these classes should not hold more than 10–15 pupils. If a child suffers from such a high degree of deafness that he cannot follow the lessons in a special class, he should be placed in an institution for the deaf and dumb, assuming, of course, that in such institutes in large cities the lessons are based on the hearing principles for children who still possess serviceable hearing remnants.

All the pedagogic arrangements which tend to promote the educational facilities of the children presuppose a well-organized medical service. The function of the school physician has occupied the foreground of school-hygienic questions for a number of years.

In June, 1908, the Austrian Society for the Education of Children sent out a question blank with reference to the school physician, the principal object of which was to establish the way a medical service in the schools of large cities could be promptly established.

The organization of the medical school service in Berndorf, in Lower Austria, is a model arrangement.

In answer to the question blank referred to, Dr. Dehne reported his experience in connection with the medical school service in Berndorf. This experience comprises a period of four years, and has shown that Dr. Dehne has instituted and carried out that service in an excellent manner.

One of the city physicians acts as school physician. Specialists are engaged for the examination of the ear, eye, and teeth. The additional engagement of an orthopædist would be desirable.

In my capacity as school otologist I undertake the examination of the ears, nose, and fauces every Saturday and Sunday during the month of May. The ear has already been included in the general physical examination made by the school physician, so far as otoscopic findings and hearing acuity are concerned, likewise the nose, fauces, and speech. He determines which children should be sent to the otologist. In urgent cases a special examination, of course, takes place immediately. Should extensive surgical measures be necessary, the child is sent by the school physician to my department at the General Policlinic in Vienna. Berndorf has an ear clinic for school children since 1909, thanks to the munificence of Arthur Krupp. It is equipped with all modern appliances.

A short history is established in each case, and I determine the hearing acuity, the tuning-fork test, and, if necessary, the functional test of the labyrinth, according to the methods of clinical tests described on p. 96.

The school otologist has also to pay the greatest attention to an examination of the nose and nasopharyngeal space, aside from examin-

ing the ear. My experience in Berndorf has shown that up to the period of puberty hypertrophy of the tonsils undergoes spontaneous involution in but a very small number of cases. It has also shown that the frequency of catarrhal middle-ear affections considerably decreases during school age. According to the statistics of 1908, children with hypertrophic tonsils between the ages of six and seven include 85 per cent. of catarrhal middle-ear affections, as against 29 per cent. between the ages of thirteen and fourteen, although the etiologically important changes (hypertrophic tonsils and adenoid vegetations) had not undergone any material change in the meantime. On the other hand, the percentage of cases with middle-ear *suppuration* in the presence of hypertrophic tonsils considerably increases during school age. As the danger of catarrhal middle-ear affections during school age decreases, the danger of suppurative otitis media increases; in fact, the decrease in the percentage of catarrhal middle-ear affections is exceeded by the increases in the percentage of suppurative middle-ear affections.

It cannot, therefore, be asserted that the danger of hypertrophic tonsils for the growing child decreases during school age; on the contrary, the danger increases. The danger in the first period of school life, from the seventh to the eleventh year, lies in catarrhal middle-ear affections, which is superseded in later years by the danger of suppurative otitis media.

It may be objected that the greater frequency of the latter affection is due to the infectious diseases which children usually acquire during that period, but it should be remembered that even in these cases the hygienic condition of the nasopharyngeal tract is the most important factor: a child with a healthy nasopharyngeal tract will, in the course of scarlet fever, measles, diphtheria, etc., not acquire suppurative otitis media as easily as one with hypertrophic tonsils, adenoid vegetations, and impaired or obstructed nasal respiration.

It follows of necessity that hypertrophic tonsils should be removed so soon as they impair normal respiration or the physiologic ventilation of the tympanic cavity.

The correctness of this statement was confirmed during an epidemic of scarlatina and measles in Berndorf in the winter of 1909–1910. Among the patients there were several children whose hypertrophic tonsils and adenoids I had removed one or two years previously: not one of these contracted an acute middle-ear suppuration in the course of the infectious diseases.

The percentage of children with permanently reduced hearing acuity increases during the school period, but this percentage can be reduced to a minimum by methodical examination and competent treatment commencing at school age.

It is necessary, in the interests of a successful activity of the school otologist, that teachers and parents be advised on hygienic questions concerning the ear. The school physician, or another suitable teacher, should deliver lectures from time to time on personal hygiene (somatology), including the hygiene of the ear. A useful arrangement for larger cities would be for the school otologist to deliver an annual lecture before the pupils collected from several schools, and their parents, on the hygiene of the ear and the nasopharyngeal tract.

The school otologist examines the children, if possible, in the presence of the school physician and the parents. The invitations to attend the examinations distinctly state that the examination is not compulsory. After the examination the school otologist makes his diagnosis and decides upon the form of treatment.

The conservative treatment is delegated to the family physician, preferably in an interview between the school otologist, the school physician, and the family physician. This interview should take place after a large number of examinations have been made. If desirable, a member of the teaching staff may also be invited to attend. The school or family physician will undertake to inform parents of the decision of the school otologist in regard to the necessity of surgical interference, and obtain their consent. It is advisable to compile the salient points concerning the hygiene of the ear in a special circular for the use of children, parents, and teachers.

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